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CONVALESCENT CARE OF PATIENTS WITH CRANIOCEREBRAL INJURIES*

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THE care of patients who are convalescing from craniocerebral injuries has been largely empirical. Little attention has been paid to working out its details, and no attempt has been made to correlate the patient's needs with the antecedent injury and the resulting pathologic lesion. After leaving the hospital, the victim is usually told to "take it easy for a few weeks or months," "not to worry" and "not to return to work until he feels up to it." Headaches, dizziness, lack of concentration, easy fatigability, and the like are considered to be the usual accompaniments of the convalescent period. If they last longer than the doctor's patience, these same symptoms then go under the name of "post-traumatic neurosis" and the patient becomes a surgical derelict. While the primary need is for a better understanding of the original lesion caused by the craniocerebral injury, later intelligent oversight of the patient and his family is also requisite if the therapy given during the acute stages is to yield the results that it should.

THE PRECONVALESCENT PERIOD

The length of the preconvalescent period will vary with the kind of injury, the accuracy of the doctor's diagnosis and the efficiency of his treatment. For example, if the patient has been diagnosed as a case of "concussion" and actually had concussion, he will have no preconvalescent period at all inasmuch as, by definition, the disease is ended with his return to consciousness. On the other hand, if the case has been diagnosed as one of "concussion" but the patient has in reality had a lacerated brain, or has been diagnosed as suffering from a "fractured skull" and really harbors a subdural hematoma, his preconvalescent care will constitute a major part of his treatment. Indeed, in the latter case it cannot be considered as having ended until after the removal of the blood clot or its fluid equivalent, a procedure which may not be recognized as essential until years after the receipt of the injury.

Broadly speaking, preconvalescence after craniocerebral injuries ends and convalescence begins when the intracranial pressure has remained fixed at normal for an arbitrary period of two weeks. It should be understood, however, that previous to the start of this period all meningeal clots have been removed, all depressed fragments of bone been elevated or removed and no extracranial or intracranial sepsis is present. Two weeks is commonly considered as adequate for any normal wound-healing, provided the local circulation is efficient and the part at rest so that organization can be effected. Surgically clean cerebral injuries or wounds have the same requirements for healing that similar wounds elsewhere in the body do. In cerebral wounds, however, the local circulation is efficient only in the presence of a normal intracranial pressure. When this pressure is high, there is an associated venous congestion and local tissue anoxia. When it is subnormal, my experience convinces me that the patient's water metabolism is affected, that he is dehydrated and that here too the local circulation is inefficient. It is on this basis that the convalescence after craniocerebral injuries is held to begin at the end of a two-week period of constantly normal intracranial pressure, the part being maintained at rest and local organization effected by keeping the patient constantly in bed. Except as the result of tissue deficit or erroneous diagnosis, the great majority of patients are symptom-free during this time. If they are not, and meningeal clots and local sepsis have been eliminated by appropriate measures, the cause of their symptomatology will usually be found in associated disease processes such as arteriosclerosis, renal disease, impaired circulation, associated brain tumors, syphilis, multiple sclerosis, other injuries and the like.

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THE CONVALESCENT PERIOD

With the wound healed and the patient free of symptoms, convalescent care after any disease is merely a matter of improving the patient's general condition in so far as his make-up and permanent physical deformities permit. Even adequate care, however, cannot be expected to correct symptoms that are traceable to actual loss or destruction of brain tissue, to the effects of scars or sepsis on the cerebrum, or to associated or intercurrent disease processes. Recognition of and adjustment to the deficits, and treatment of the associated and perhaps unrelated pathologic lesions, are easier and more efficient, however, if the convalescent care of the fundamental damage is intelligent and suited to the patient's needs and understanding.

Adequate but not too much rest, and constantly increasing exercise are classic, efficient and reliable methods for accomplishing this end. This holds true for cerebral as well as for any other wounds. However, the method of application differs in the former because of the curious psychologic reaction that the patient and his relatives and friends have toward injuries to the head or brain. As applied to the patient, this takes the form of a loss of initiative and unwillingness to accept responsibility for his own decisions. As applied to the family and friends, it takes the form of a supersolicitude which is fatally enervating and which supplements the patient's loss of initiative. If these factors are overlooked and not counteracted, the patient does not exercise except when it gives him pleasure, his rest is increased out of all proportion to his needs and his general physical condition not only does not improve but actually deteriorates. His relatives and friends and often his doctor are expected to wait on him more and more. This saps his initiative still further. It does not take much of this to produce the habit of invalidism from which it is only a short step to a permanent neurosis, the development of a new subjective symptomatology to rationalize this neurosis and permanent loss of earning power, self-respect and character. Once this succession of events has fairly started, it is almost impossible to interrupt its progress. Our only hope of reducing the numbers of such permanent invalids following craniocerebral injuries lies in its prevention.

The essential elements — the loss of initiative and the associated lack of self-confidence — are apparently due to two factors. One is the conviction in the average person's mind that a brain injury necessarily means insanity, epilepsy, loss of memory and judgment, and permanent invalidism. This, I should suppose, has its roots in the ideas that recognized evil spirits and witchcraft. In line with the medical education that teaches us to go to bed and stay there in case of serious illness, the patient, in an attempt to avoid these aftermaths, will naturally tend to reduce his activities to the minimum unless urged to do otherwise. Another factor is a loss of confidence in the medical profession. This is due to the fact that it is common knowledge among the laity that doctors not only cannot cure but do not even believe in the reality of symptoms that persist after craniocerebral injury. An individual who is suffering from persistent headaches and who is honestly unable to earn his living on account of his incapacity is not to be put off with platitudes by doctor after doctor and still be expected to keep his confidence in the medical profession. Any well-managed convalescent regime after craniocerebral injuries should be so planned as to eliminate all element of "magic," and so as to ensure the retention by the doctor of the patient's confidence.

Convalescence after craniocerebral injury may be summed up, then, as a period during which an individual whose wounds have just healed needs direction in regard to returning his general physical condition to normal or better. These directions must be conditioned, however, by the doctor's recognition that the most important element in preventing the attainment of this end will be the patient's loss of initiative and lack of self-confidence. Any plan for convalescent care must, therefore, not only provide directions as to physical activity but must embody in them enough applied psychology to prevent the patient from interfering with his own recovery.

The first increase in physical activity comes with the start of the convalescent period, when the patient gets out of bed. This is the first opportunity to prevent him from losing his initiative. To accomplish this, he is directed to get out of bed in accordance with his own schedule. Specifically, the nurse and he are both made to understand that he and he alone decides how long he shall stay up and how long he shall lie down. He is told to govern the length of these periods by the way he feels. Thus, he is to stay up until he is tired, then return to bed and stay there until rested and then get up again. This start is important. It forces the patient to accept the responsibility for his own actions at the earliest possible moment. If he overdoes and develops symptoms, it is his fault; if he is underactive and does not progress, again it is his fault. Alibis and hence the development of rationalizing symptomatology are nipped in the bud.

As his activity increases, he begins to ask about leaving the hospital. This provides the doctor with the second opportunity to counteract loss of in-

itiative. Serious illness in the home is invariably accompanied — in the lay mind at least — by partial to complete limitation of the patient's activity to one floor and the taking of perhaps all meals but certainly breakfast in bed. It is equally true that it is impossible to look on an individual as seriously ill or as an invalid when he goes up and down stairs freely and has, as a matter of course, all his meals in the dining-room with his family. If the patient can be made to accept, without suspicion, the hospital equivalent of complete activity about his home, the chance of his developing invalidism in these latter surroundings is greatly decreased. However, all the patient knows at this time is that he is up and about all day and wants to go home. When told that his return home depends on his ability to remain active all day and to take two flights of stairs twice in succession he proceeds to do so at once. Thus, he has been persuaded to demonstrate publicly that he is neither physically incapacitated nor an invalid. Failure to live up to his own specifications after his return to the community labels him as a fool, a liar or both, and alienates what would otherwise prove to be the harmful sympathy of his family and friends.

Before letting the patient leave the hospital, however, he and his nearest relative are told in detail what permanent defects have been produced by his injury. It is important to do this at this time, and it is important to include a member of the family in the audience because only in this way can claims to invalidism based on alleged late discovery of a disabling symptom be forestalled. At this same interview it is also explained that the patient's further recovery depends solely on his improvement in general strength, that he is the only person who can bring this about and that he can only do it by taking increasingly larger amounts of regular exercise interspersed with adequate rest. He is further assured that if he gets overtired he will have symptoms due not to his brain injury, which is healed, but to exhaustion; and that if he loafs he will remain an invalid. A simple set of directions is given him. These call for out-of-door exercise every morning and afternoon, nine or ten hours in bed every night, a rest period on his back for one hour before the midday and evening meals, regular hours, plenty of water and no medicine. There are three prohibitions. The first is that he must never again drink alcohol in any form. It has been found that alcohol taken after craniocerebral injuries predisposes to convulsions and is abnormally toxic. The second is that he must not dive head-first into water. It is believed that the sudden change from air to water pressure as applied to the head with the rest of the body still in the air will frequently cause the patient to faint, with resultant drowning. The third prohibition is not to go onto high places unless there is provision against falling off if he becomes unconscious. Here, too, it is believed that many such patients are more than usually apt to faint under such conditions.

With these directions, the patient is sent out "on his own" for a month. This is the period that makes or breaks him, especially if he is a manual laborer. The ones in this group, regardless of the original injury or its treatment, especially if they belong to the emotionally labile races like the Italians and Irish, are the most likely to be failures. During this first month of convalescence, they, like all who are injured by other people's carelessness or while at work, are subjected to influences that have as their object not the promotion of the physical welfare of the victim but rather his cash value. Insurance adjusters, lawyers, police, interested friends and relatives, even interested doctors, begin to urge him to develop more symptoms, accuse him of malingering or refuse to accept as genuine symptoms that he knows are real. The development of a neurosis, under such circumstances, must be looked on as inevitable and a matter of self-protection. If, because of his economic situation, he tries to go back to work, he finds that he is expected to return to his original job whether or not it is suitable, and furthermore must start in on a full-time basis. If he is unable to do this, he lays himself open to the accusation of loafing and shirking. I do not mean to imply that human nature as represented by the injured working man has any fundamental difference from that as represented by the stockbroker, the lawyer or even the doctor. There is always a certain irreducible minimum of the population who will seize any opportunity and use any means to feather their own nests. I do believe, however, that the percentage of such individuals among patients with late symptoms after craniocerebral injuries is far lower than is generally conceded.

If the patient gets through this first month of his convalescence successfully and reports back to his doctor for further instruction, he should be urged to increase his exercise, diminish his rest period and carry on for another month. At the end of the second month, he should be able to return to work on a part-time basis in a job that is suited to his new physical capacity and, at the end of another two weeks, be a full-time employee again. Furthermore, he should have learned by then that his craniocerebral injury is actually a thing of the past, that his convalescence did bring his general condition back to normal and that

his initiative and self-confidence are as good as they were before his injury.

It can doubtless be successfully argued that there have been many patients with craniocerebral injuries who have fully recovered without such detailed convalescent care. I am also fully aware that recoveries even with this regime are not so common as I should like or as is desirable. As long as lacerated brains are diagnosed as concussion, and subdural hematomas go unrecognized for years, attempts to estimate the effects of any method of treatment on the end results of acute craniocerebral injuries are futile. Nevertheless a start must be made, and a therapy, no matter how bad it is, that includes planned care during convalescence is surely better than one that ignores this stage of the illness altogether.

SUMMARY AND CONCLUSIONS

The convalescence of a patient who is recovering from a craniocerebral injury should be planned in such a way as to correlate his needs with the antecedent injury and the resulting pathologic lesion.

The preconvalescent period may vary from a minimum of seconds in a properly diagnosed case of concussion to years in a neglected case of unrecognized subdural hematoma.

The convalescent period begins after the patient has had a constantly normal intracranial pressure for two weeks.

Loss of initiative and lack of self-confidence on the part of the patient are constant accompaniments of a craniocerebral injury. Otherwise such injuries differ in no important way from wounds elsewhere in the body.

The convalescent care of patients with craniocerebral injuries should not be of a hit-or-miss type, but must be carefully planned to keep this associated destruction of initiative and self-confidence from developing into a permanent neurosis and spoiling the end result of what might otherwise have been a successful case.

TECHNIC FOR THE SUCCESSFUL USE OF PROTAMINE–ZINC INSULIN*

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A TIMELY editorial in a recent number of the *Journal of the American Medical Association*[1] calls attention to the fact that all is not so well with the use of protamine-zinc insulin, and that the untoward effects of this agent are becoming steadily more recognized. In a paper describing the advantages and disadvantages of protamine-zinc insulin one of us (W. S. C.)[2] called specific attention to some of its undesirable features and recommended certain precautionary measures in its employment.

This paper is concerned with the presentation of certain recommendations which will aid in taking full advantage of the prolonged effect of precipitated insulin, and at the same time avoid the pitfalls and complications attendant on its use.

Before presenting our recommendations, it is well to review the pharmacologic properties of unmodified insulin and protamine-zinc insulin, especially in relation to their effect in lowering the blood sugar. The injection of a dose of unmodified insulin produces certain characteristic changes in the blood-sugar curve. If it is given intravenously there occurs a refractory period of almost ten minutes during which the blood sugar remains at a practically stationary level. At the end of this period there occurs a very rapid and precipitous drop in the blood sugar. Regardless of the dose, we have observed that the rate of fall in blood sugar is roughly 1.7 mg. per unit per minute. The total drop is somewhat but not greatly influenced by the dose of insulin administered. The important difference between a large and small dose lies in the total duration of the insulin effect. The larger the dose, the longer will its effect be manifested. These results are similar in the diabetic patient whose initial blood sugar is abnormally high or low. The use of unmodified insulin by the subcutaneous route produces blood-sugar curves which in their general configuration resemble those following the intravenous administration of insulin.

Protamine-zinc insulin, on the other hand, produces a decided change in the character of the action described above. By virtue of the fact that this precipitated insulin is slowly soluble in blood serum, it obviously is not so promptly available as is the unmodified insulin. Enough experimental investigations of the blood-sugar curve following

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the administration of protamine-zinc insulin have been reported[3] to indicate that its full effect is not manifested before the seventh or eighth hour after its administration.

Because of the slow liberation of the molecule of insulin from its precipitated form, the duration of the insulin effect becomes definitely prolonged. Thus as it is at present commonly employed, protamine-zinc insulin, in order to be made therapeutically effective during the prandial state, must be given in a dose so large as to be effective during the prandial period of the following day. The chief difficulty in the use of protamine-zinc insulin appears to lie in the fact that the lasting action of the drug has the effect of producing a hypoglycemic state during the postabsorptive period, which lies between 2 a. m. and 8 a. m. This has been borne out by our own clinical experience that the commonest period during which patients have hypoglycemic attacks from protamine-zinc insulin occurs between four and seven in the morning.

It is well in passing to state that the clinical picture of insulin overdosage following the use of protamine-zinc insulin is vastly different from that produced by unmodified insulin. The former is usually characterized by manifestations associated with functional disorders of the central nervous system, such as drowsiness, headache, listlessness, diplopia, asphasia, fatigue, nausea, paresthesias and marked emotional crises, such as crying spells, shrieking, transitory depressions and coma. The well-known shock symptoms which follow overdosage of unmodified insulin such as trembling, pallor, sweats and hunger seem to be curiously absent.

The experience of a hypoglycemic attack must be looked upon as a serious and harmful episode, notwithstanding its occasionally trivial and transient character. Baker and Lufkin[4] have observed multiple petechial cerebral hemorrhages in animals that died following a long-standing insulin hypoglycemia. Many reports in the literature have seemed to prove that insulin shock in the elderly diabetic patient is attended by such vascular effects as spasm or thrombosis of cerebral arteries and coronary arteries.[5] Pathologic changes in the electrocardiogram have been reported during periods of insulin hypoglycemia.[6]

We must at this point call attention to an extremely important phenomenon in connection with hypoglycemic states. In the investigation of fasting blood-sugar determinations on patients treated with protamine-zinc insulin, we have not infrequently observed blood sugars as low as 40 mg. per 100 cc. in a patient entirely without symptoms. The dangers in the use of protamine-zinc insulin thus become obvious if the hypoglycemic state can exist in an individual free of subjective or objective clinical manifestations of this condition.

From our experience with the use of protamine-zinc insulin in 375 diabetic patients over a period of almost two years, we have established a technic which we find to be successful. With our procedure we are able to secure the maximum efficiency of the agent and at the same time avoid untoward reactions. It is our belief that many reports in the literature which have a tendency to discredit the clinical value of protamine-zinc insulin are due to a faulty technic.

TECHNIC

There are several factors to be considered in the proper use of protamine-zinc insulin. They are as follows.

Selection of Patient

If a diabetic patient is receiving one dose of insulin a day, is living on a diet sufficient to meet with the physiological requirements, is free of sugar in his urine and his blood sugar is close to the normal range, we consider him adequately controlled and not a candidate for protamine-zinc insulin. When, on the other hand, the diabetes is severe enough to warrant the use of two or more doses of insulin a day, protamine-zinc insulin should be employed. This statement, however, is made with a slight reservation. Since the elderly, sclerotic diabetic patient is susceptible to serious clinical complications arising from vascular spasms, we think that the hypoglycemic state in these cases must under all circumstances be avoided. Thus the elderly diabetic patient is generally not a suitable subject for protamine-zinc insulin.

In summary, we find that the patients in whom protamine-zinc insulin produces the best results are those between the first and fourth decades with severe diabetes requiring more than one dose of insulin a day.

Insulin

Our method of using protamine-zinc insulin is based on the following principle. Since several hours elapse after the injection of the drug before its pharmacologic action becomes manifest, and since its effective use comes during the postabsorptive period of the same day and the prandial state of the following day, we have found that the safest way in which to use protamine-zinc insulin is to resort to the administration of unmodified insulin for the production of the insulin effect in the prandial state, and to depend on protamine-zinc insulin for its blood-sugar-lowering effect during the postabsorptive state. Stated in different terms, twenty-four hours after the administration of

protamine-zinc insulin the patient should be recovering from an insulin effect. In order to carry this out in practice it becomes necessary to aim to give minimal doses of protamine-zinc insulin, and to resort to the use of unmodified insulin for the care of the immediate prandial period. We have found in practice that when a patient's total insulin requirements are greater than 20 units a day, an additional injection of unmodified insulin should be given. In the successful use of insulin in our cases, producing maximum insulin efficiency with minimal episodes of hypoglycemia, we cite the following typical combinations of doses which different patients take:

PROTAMINE-ZINC INSULIN	UNMODIFIED INSULIN
units	*units*
20	5
20	10
25	10
25	15
30	15
40	15
40	20
40	30
40	40
50	40
60	40

In other words, the dose of protamine-zinc insulin best suited to most of our patients varies between 20 and 60 units, depending on the severity of the disease. If the patient needs more than 20 units, the additional use of unmodified insulin is resorted to. The dose of the latter varies between 5 and 40 units. The preferable time to administer either or both types of insulin is in the morning before breakfast.

It is important to instruct the patient not to mix the unmodified and protamine-zinc insulin when both preparations are to be taken. They should be given in two separate injections, one following the other and in different sites. The reason for this is that, since insulin is precipitated by protamine in an alkaline state, and the reaction is so adjusted in a protamine-zinc insulin preparation, its addition to commercial unmodified insulin, which is an acid solution, has the effect of returning it to a soluble form, thus vitiating the benefits of the precipitated insulin.

DIET

The determination of the diet from the standpoint of its total caloric value or its various components of carbohydrate, protein or fat is beyond the scope of this paper. The important consideration in the diet in relation to our suggested method for the employment of protamine-zinc insulin is that half the total carbohydrate should be divided between breakfast and lunch. The other half should be distributed between the evening meal and the intercibal feedings. In practice we find that the following proportions are optimal: breakfast, 25 per cent; lunch, 25 per cent; dinner, 20 per cent; intercibal feedings, 30 per cent.

We stress the use of intercibal feedings as being one of the most important factors in the proper management of the diabetic patient receiving insulin therapy. These feedings when properly timed have the effect of furnishing the patient with carbohydrate at a time when he is under a maximum insulin influence and is susceptible to hypoglycemic attacks. Knowing that unmodified insulin has an immediate and profound effect, patients are very susceptible to hypoglycemic attacks two or three hours after its injection. Thus the administration of 15 gm. of rapidly absorbable carbohydrate in the form of orange juice or any other fruit given two hours after the insulin protects the patient against a hypoglycemic attack. Lunch should be given exactly four hours after breakfast, followed by another intercibal feeding about three hours later. A third such feeding should be given just before retiring, and should consist of carbohydrates and also some protein from which the carbohydrates are slowly made available. Thus a practical feeding schedule consists in the distribution of the carbohydrates in the following way:

TIME	MEAL	PER CENT OF TOTAL CARBOHYDRATE IN 24 HOURS
8 a. m.	Breakfast	25
10 a. m.	Intercibal feeding	7
12 noon	Lunch	25
3–4 p. m.	Intercibal feeding	7
6 p. m. (later, if possible)	Dinner	20
11–12 p. m.	Postcibal feeding	15

FOLLOW-UP CARE

The subsequent management requires that the patient examine his urine every morning on arising and every evening before dinner. Visits to the physician should be made at monthly intervals. The patient should come prepared with a fractional collection of urines of the previous twenty-four hours. This is a great advantage over the old method of examining a mixed twenty-four-hour sample. The investigation of the fractional urines will disclose the time when the patient is excreting sugar and the time when he is not. The different time periods in the pharmacologic action of protamine-zinc insulin and unmodified insulin being known, it is obvious that the period of the insulin effect should be established with fractional urine examinations. These fractional specimens need not have any relation to the feedings, but should be those that the patient voluntarily voids at all times in the twenty-four-hour cycle before his visit. The fasting blood sugar should be determined at this time. The information obtained from both the fasting blood sugar and the fractional twenty-four-hour specimens of urine will aid materially in determining any alteration in the

dose of insulin. The dose of protamine-zinc insulin is changed according to the blood-sugar findings. An elevation in the blood sugar means that the patient is receiving an insufficient quantity of protamine-zinc insulin. A hypoglycemic blood sugar means that the patient is receiving too much protamine-zinc insulin. The dose of unmodified insulin, on the other hand, is altered depending on the character of the fractional urine estimation. If there is prandial glycosuria, there is of course an indication for an increase of the unmodified insulin. The absence of prandial glycosuria with prandial hypoglycemic attacks requires a reduction in the unmodified insulin.

TREATMENT OF HYPOGLYCEMIA

Hypoglycemic reactions occurring during prandial periods will develop in the intercibal periods if the patient is either tardy with the intercibal feedings, entirely neglects to take them or is receiving too much unmodified insulin. These reactions are easily controlled by means of an immediate feeding of a soluble carbohydrate such as 120 cc. of orange juice to which two teaspoonfuls of sugar have been added. The patient will then be completely relieved of his attack and will have no further difficulty, for his feedings will protect him against an immediate recurrence. The picture, however, of hypoglycemia which occurs during the night is vastly different, for it is the result of the action of protamine-zinc insulin. The immediate administration of rapidly absorbable carbohydrate will have the effect of relieving the attack, only to have the hypoglycemic symptoms return within an hour or an hour and a half. This is because the carbohydrate has already been consumed and the protamine-zinc insulin is still producing its profound action. The treatment of the protamine-zinc insulin hypoglycemia should in this case consist of feeding rapidly absorbable carbohydrates every hour after the attack until the physician is certain that the patient is over his insulin effect. This may last as long as from four to six hours. Under these circumstances all insulin should be discontinued until traces of sugar have appeared in the urine.

TREATMENT OF BROKEN CARBOHYDRATE TOLERANCE

It is a well-known fact that the development of any infection in a diabetic patient is characterized by a marked reduction in insulin efficiency and results in a rapid break of his carbohydrate tolerance. This peculiar vulnerability of insulin activity to the presence of infection appears to be more marked in cases where protamine-zinc insulin is employed. This inactivation of protamine-zinc insulin by infection has been experimentally demonstrated by Himwich and Fazekas[7] and we have observed the same phenomenon clinically. Where the break in tolerance becomes severe, it is advisable to suspend temporarily the use of protamine-zinc insulin and resort to multiple injections of unmodified insulin until the period of infection has passed. If, on the other hand, the break in tolerance is only mild, adequate control can be established by increasing both modified and unmodified insulin in the morning and giving an additional dose of unmodified insulin before the evening meal. It is advisable that fractional urines be examined during the entire period of the break in tolerance.

TECHNIC FOR TRANSFERRING THE PATIENT FROM UNMODIFIED INSULIN TO PROTAMINE-ZINC INSULIN

There appears to be considerable fear in the minds of many physicians regarding the transfer of patients who have been on a regime with unmodified insulin to one of protamine-zinc insulin. Observers have frequently recommended a period of hospitalization in order to effect this transfer. We do not find this procedure necessary under any circumstances. The transfer can be easily performed in ordinary office practice. If a controlled patient is taking up to 20 units of unmodified insulin a day in divided doses, it is possible to give him 20 units of protamine-zinc insulin in one dose before breakfast. If, on the other hand, his diabetic control is established with over 20 units of insulin a day, we recommend the additional employment of unmodified insulin. Transfers may be effected according to the following schedule:

Previous Total Dose	New Dose	
Unmodified Insulin	Unmodified Insulin	Protamine-Zinc Insulin
units	*units*	*units*
20	0	20
30	5	20
40	10	25
50	15	30
60	20	30
70	25	35–40
80	25	40–50
90	30	40–50
100	35	50–60

It is important to remember that the dietetic suggestions which we have recommended, especially in so far as the use of intercibal feedings is concerned, should be rigidly followed. It is suggested that fractional urine samples be examined daily for approximately one week before the stabilizing dose of protamine-zinc insulin is established. The fasting blood-sugar level should be determined once a week for the first month after the transfer.

CONCLUSIONS

A technic for the successful employment of protamine-zinc insulin is described, whereby it is

possible to exploit all the benefits of protamine-zinc insulin and at the same time prevent its undesirable side effects. We recommend the use of this agent as a far-reaching advance in the treatment of diabetes.

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CHIARI'S SYNDROME IN A PATIENT WITH POLYCYTHEMIA VERA*

Report of a Case

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OCCLUSION of the hepatic veins due to thrombophlebitis is uncommon, but when it occurs it produces a well-defined group of signs and symptoms. Although the earliest recorded case of this symptom-complex is that of Budd,[3] Chiari's[4, 9] more complete studies in 5 patients seen in his clinic forty to fifty years later have led to the connection of his name with the syndrome. As has been pointed out in the reviews by Hess,[9] Thompson and Turnbull,[19] Satke[16] and Hutchison and Simpson,[10] the hepatic thrombophlebitis responsible for the clinical picture may be either primary or secondary to a variety of lesions situated at the junction of the hepatic veins and inferior vena cava. These lesions include neoplasm, hydatid cyst, gumma, nonspecific inflammatory masses, chronic perihepatitis and cirrhosis of the liver.

The occurrence of this symptom complex in polycythemia is extremely rare. One case was described by Oppenheimer[15] in 1929; since then 7 others have been reported.[2, 5, 12–14, 17, 20] Of the 8 cases of the syndrome thus far described, the hepatic thrombophlebitis occurred alone in 5,[2, 12–15] in association with thrombophlebitis of other abdominal viscera in 2[5, 17] and as a complication of cirrhosis of the liver in 1.[20]

Because of the rarity of the syndrome, it has been thought desirable to report the following case.

CASE REPORT

C. S. (B.I.H. 41557), a 27-year-old American white married painter, entered the Beth Israel Hospital on May 20, 1938, complaining of swelling of the legs of 1 week's duration. The family history was negative except for tuberculosis in one brother. The past history revealed the usual childhood diseases, frequent head colds and an episode of diarrhea lasting several days 3 years before admission. The present illness began 3 weeks before entry with sudden onset of dizziness, epigastric distress, a sensation of warmth and malaise. A physician told the patient he had the grippe. His fever and malaise subsided in 3 or 4 days. A week before admission he developed severe pain in the flanks which lasted for about a day. Three days later his abdomen and ankles became swollen.

Physical examination on admission was negative except for dullness and diminished breath sounds and fremitus at the right lung base posteriorly, shifting dullness and fluid wave in the abdomen and moderate pitting edema of the legs. Ophthalmoscopic examination showed only moderately engorged veins. The blood pressure was 115/80.

The red-blood-cell count fluctuated between 6,760,000 and 6,890,000, with a hemoglobin of 121 to 132 per cent (Dare). The white-blood-cell count ranged between 11,000 and 21,000, with 71 to 91 per cent polymorphonuclears. The hematocrit was 64 per cent, and the platelet count 2,000,000. Four urine examinations revealed a maximum specific gravity of 1.024. All other urinary findings were negative except for the presence of bile. A small amount was noted on the day of admission; this increased during the next week. Five stool examinations were not remarkable. The blood nonprotein nitrogen was 45 mg. per 100 cc. on admission, fell to 35 mg. 2 days later and then rose to 100 mg. The icteric index on admission was 6 and rose steadily to 30, at which time the serum bilirubin was 4.2 mg. per 100 cc. The serum protein concentration was 5.8 gm. per 100 cc. The serum cholesterol was 135 mg. per 100 cc. on two occasions, with a cholesterol-ester value of 37 mg. Blood Hinton and Kahn reactions were negative. The venous pressure was 10 cm. by the direct method, and the arm-to-tongue decholin circulation time was 12 seconds. A galactose-tolerance test revealed excretion of 1.03 gm. after the ingestion of 30 gm. The total extracellular fluid volume on May 23, as calculated by the method of Crandall and Anderson,[6] was 24.7 l., a value approximately 40 per cent higher than normal for his body build.* An electrocardiogram on May 25 was normal.

X-ray examination of the heart and lungs was not remarkable except for elevation and diminished respiratory movement of the right dome of the diaphragm.

The pitting on pressure of the tissues of the legs was not elicited after the 2nd hospital day. An abdominal

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*The actual excess water available for the solution of sodium thiocyanate was 6.6 l.; this includes the total excess extracellular fluid and the excess fluid of the blood. Subtracting the probable excess fluid in the blood due to the hypervolemia of polycythemia, the figures indicate approximately 6.0 l. of edema fluid.

paracentesis was performed on May 23, with the removal of 1800 cc. of clear yellow fluid. The specific gravity of the fluid was 1.015 and the protein content 2.8 gm. per 100 cc. Following paracentesis the liver was found to be tender and enlarged several centimeters below the costal margin. The spleen was also palpable 3 cm. below the costal margin. Two days later painful tender nodules were noted along the course of the veins of the right calf. Distended veins appeared over the lower thorax anteriorly and the upper abdomen. At this time a visiting physician suggested the diagnosis of thrombophlebitis of the iliac veins, of the inferior vena cava below the level of the kidneys and of the portal vein, all probably associated with polycythemia vera. The temperature, which had been normal during the first 7 days, commenced to rise. The patient lost all desire for food, commenced to vomit, developed moderate jaundice and rapidly became comatose. He died on the 9th day.

Postmortem Examination (A-38-46). This was performed half an hour after death. The peritoneal cavity contained approximately 500 cc. of clear, deeply bile-stained fluid. The liver was slightly enlarged, the lower edge extending 2 cm. below the costal margin; it weighed 2140 gm. The greater portion of the right lobe was swollen and poorly demarkated. The caudate lobe was anomalous, appearing as two curved finger-like masses. There were some old fibrous adhesions between the under surface of the right lobe and the duodenum; elsewhere the capsule was smooth and glistening. Sections through the liver revealed the previously described swollen portion of the right lobe as consisting of a fatty yellowish tissue poorly demarkated from the normal brownish-red liver tissue in the rest of the lobe. The entire caudate lobe showed the same swelling and fatty changes, but the left lobe was apparently not involved in the degenerative process. Approximately three quarters of all the liver tissue shared the degenerative changes grossly. In the right lobe, a little below the capsule, there were found two irregular, sharply defined firm, yellowish-white, smooth, somewhat hyaline masses, each about 2.5 by 1 by 1.5 cm. These appeared to be healed infarcts. The hepatic veins in the entire right and caudate lobes were thrombosed, all the small and medium-sized branches being involved. The thrombi in the smaller peripheral veins completely occluded the vessel lumens, and were adherent to the walls. All were red, and in most cases soft in consistence; a few were firm and apparently organizing. In the larger, more proximal veins the thrombi were a deeper red and soft. Although most of these larger vessels were completely occluded by the thrombi, an occasional vein showed only partial occlusion with thrombi which were not firmly attached to the vessel wall. The thrombotic process was continuous from the smaller vessels to the entrance of the hepatic vein into the vena cava, a small thrombus occurring also in this latter vessel, but not occluding it. The thromboses in the right lobe were situated in areas of necrosis and in the remaining normal-appearing areas. None were found in the left lobe of the liver. The intrahepatic and extrahepatic bile ducts were not remarkable. The branches of the portal vein within the liver were entirely free of thrombi and not unusual.

There was marked dilatation of many of the branches of the portal veins outside the liver, particularly those in the mesentery of the small intestine. Many dilated veins were also found on the peritoneal surface of the anterior abdominal wall. The inferior vena cava was not dilated and, except for the area about the insertion of the hepatic veins, was free of thrombi.

The spleen was greatly enlarged, weighing 980 gm. It was dark purplish-red and firm in consistence. Sections through the spleen revealed a congested pulp.

The other gross findings consisted of congestion of the lungs and a deep purplish-red, cellular bone marrow.

Many sections taken from representative portions of all lobes of the liver, including the areas which grossly appeared to be unaffected, revealed essentially the same process, varying only in intensity. The process consisted of central necrosis and degeneration, so extensive in some areas that the necrotic areas were confluent, leaving only scattered small areas of relatively normal liver cords around the portal spaces. In the zone between the necrotic and intact areas, the liver cords showed degenerative changes with granular deposition and vacuolization within the cell cytoplasm. Inflammatory cells, predominately lymphocytes and some polymorphonuclear neutrophilic cells, infiltrated these areas of necrosis, and slight hemorrhage was present in a few places. The stroma was better preserved and, in some lobules, showed condensation, bringing the portal areas in closer proximity than is usually found. Fibrosis was noted in only one area. The capsule of the liver was irregular and slightly thickened, with slight fibrin deposition and a few red blood cells on the surface. Bile pigment was found in the canaliculi, the latter showing no evidence of distention. Pigment deposits were also noted in the areas of necrosis. Most of the central and large hepatic veins in all the sections of liver were completely thrombosed. In scattered areas where hepatic veins were seen to be giving off branches it was apparent that the thrombi in the large vessels were older and appeared to be propagating themselves into the small veins. The extent of the liver necrosis varied as the number of vessels thrombosed. Some of the thrombi appeared to be loosely bound to the vessel wall by fibrin meshes, while many showed advanced states of organization with endothelial proliferation; most of them were made up of red blood cells, a few white blood cells and fibrin. There was a notable absence of platelets, and no lines of Zahn were noted. While the majority of the thrombi were of the red variety, a few of the mixed type were noted. The walls of the veins involved were edematous, infiltrated with fibrin and contained variable numbers of inflammatory cells. The portal veins, in contrast, were free of thrombi, definitely dilated, and in most instances filled with serum containing a few cells. A few of the portal veins were packed with red blood cells. The principal lesion consisted of widespread thrombosis of the hepatic veins and small tributaries, with extensive necrosis of the liver substance.

The lungs showed generalized vascular congestion. Small patchy areas of pneumonitis were scattered about, while many small bronchi were filled with pus. Areas of emphysema adjoining small atelectatic patches were found near the pleural surfaces. All sections of the lungs contained a few small veins and an occasional artery which showed thrombosis, in various stages of organization; most of them were, however, recent. Only one of these thrombi showed recanalization. Some of the small arteries showed endarteritis.

The spleen displayed marked congestion with increased fibrous tissue in the pulp. A slight eosinophilia was present. The kidneys exhibited the earliest stages of arteriolar sclerosis, with hyaline patches noted in the walls of the afferent arterioles. The kidneys and adrenals were congested. The prostate showed hyperplasia of the epithelial elements. The bone marrow had an increased number of islands of normoblasts; a few collections of myeloblasts were also noted. The other organs were all essentially negative.

DISCUSSION

The clinical picture of thrombophlebitis of the hepatic veins is fairly well defined. Thompson and Turnbull[19] have divided the cases into two main groups. In some patients the onset is gradual after a period of premonitory epigastric pain. Ascites and a large tender liver develop, the former recurring after repeated paracenteses, and the superficial veins of the chest and abdomen dilate. Vomiting is common. Jaundice is not prominent. Death ensues in one to six months from slowly progressive hepatic failure. In other cases the course of the disease is shortened to a week or less. Coma and delirium are of frequent occurrence in this group of cases. Rarely, life is prolonged for many years. Hutchison and Simpson[10] described a patient who lived a fairly comfortable life for twenty-five years after the apparent onset of primary hepatic vein thrombosis, dying after exploratory laparotomy.

The development of splenomegaly in this syndrome is due to changes in the dynamics of hepatic flow following hepatic vein thrombosis; these probably also contribute to the development of hepatomegaly. In the normal individual the hepatic artery and portal vein are both afferent vessels, and the hepatic veins efferent. Following closure of the hepatic veins, however, the normal egress of blood from the liver is prevented. Marked portal congestion ensues, with the appearance of splenomegaly and dilated abdominal veins. Some portions of the portal system may actually take over the efferent function. The enlargement of the liver is due largely to engorgement with blood, for at autopsy, after the blood has run out of it, the liver has almost uniformly been found much smaller than normal. In the case here reported, however, a considerable amount of swelling was due to degenerative changes in the liver parenchyma. It is possible that if the patient had lived longer the liver might have become markedly atrophied as in most other reported cases. A similar sequence of events occurs in acute hepatitis.

This syndrome may be confused with cirrhosis of the liver, acute hepatitis or thrombosis of the splenic vein. It is distinguished from the first in many cases by the rapid onset, and in most by a tender, enlarged liver. By the time the spleen becomes considerably enlarged in cirrhosis of the liver, the latter organ is usually quite small and rarely tender. The absence of marked jaundice differentiates Chiari's syndrome and acute hepatitis. Splenic-vein thrombosis may be differentiated by the occurrence of left upper-quadrant pain, frequently of a severe degree. The liver is usually not enlarged and is rarely tender. In the case here reported the finding of evidence of thrombophlebitis in the legs led to a diagnosis of visceral thrombophlebitis, although the exact site of these internal thromboses was not recognized during life. The early prominence of edema of the ankles in our case was probably due to thrombophlebitis of the veins of the legs.

The widespread occurrence of thrombotic processes may have been associated with the extremely high platelet count found in this patient with polycythemia vera, although it is to be noted that the thrombi contained only a very few platelets. It is impossible to define exactly the sequence of events which led to the development of extensive hepatic-vein thrombosis. It is possible that some localized process in the liver resulted in a purely local thrombosis which, in the presence of a greatly increased tendency toward clotting associated with the blood changes and also the slow blood flow of polycythemia, may have rapidly propagated itself to adjacent areas. As successive portions of the liver parenchyma were involved, secondary centers in which venous thromboses were occurring and from which they were spreading may have been established. It is of interest in this connection that one week before admission to the hospital the patient experienced severe pain in the flanks, possibly due to the development of the infarcts found at autopsy. Three days later ascites appeared and a week later jaundice and acidosis, as the hepatic-vein thrombosis spread and parenchymal degeneration developed.

Although the patient died before chemical studies of the blood could be completed, certain changes were noted. Diminished total cholesterol and cholesterol-ester contents of the blood were found, as in the case described by Sohval,[17] even before the development of clinically appreciable jaundice. These are to be ascribed, as pointed out by a number of authors,[1, 7, 8, 18] to widespread liver-cell damage. The rise in blood nonprotein-nitrogen concentration is to be ascribed to the development of the toxic nephritis commonly seen in patients with jaundice and severe liver damage. Jacobson and Goodpasture[11] noted the rapid development of severe acidosis in a case of thrombosis of the hepatic veins observed by them.

The changes in the liver which occur in cases with Chiari's syndrome resemble those found in the liver in those with severe congestive heart failure. These changes consist in marked engorgement of the sinusoids of the liver, with degeneration and necrosis of the liver cells in the central portion of the lobule. The involvement of each lobule in the degenerative process is much more extensive in Chiari's syndrome than it is in congestive failure, so that, as in the case reported

here, the hepatic picture approaches that of acute yellow atrophy. Most patients with thrombophlebitis of the hepatic veins do not live long enough to develop extensive fibrotic changes in the liver. The patient reported by Hutchison and Simpson,[10] however, lived twenty-five years after the apparent onset of the thrombotic process in his hepatic veins and at autopsy showed central or cardiac cirrhosis of the liver.

SUMMARY

Chiari's syndrome, thrombophlebitis of the hepatic veins, may be primary or secondary to a variety of conditions, including, as in the case reported here, polycythemia vera. The clinical, blood chemical and pathological findings are described, and the differential diagnosis discussed.

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NATIONAL AND STATE PROGRAM FOR TUBERCULOSIS CONTROL*

Presidential Address

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NOTABLE progress has recently been made in arousing national interest in the adoption of measures for the prevention and control of tuberculosis. Before discussing these matters, it is desirable to consider the problem as it presents itself in the country as a whole.

TUBERCULOSIS AS A PUBLIC-HEALTH PROBLEM

There has been a consistent decline in the death rate from tuberculosis in the United States from nearly 200 per 100,000 population in 1900 to a probable rate of below 50 for the year 1938. During 1936, for which returns[1] are available, there were 71,527 deaths from tuberculosis, a rate of 55.7 per 100,000 population. With this number of deaths, it may be estimated that there were at least five times this number of living persons with the disease. It ranked seventh among the leading causes of death without regard to age groups, and first in the most useful and productive period of life, from fifteen to forty-five years of age.

Tuberculosis control is an expensive public-health project. Considering only institutional provision for the disease, the replacement value of land, buildings and equipment for sanatoriums, tuberculosis departments and preventoriums in the country is estimated by the Council on Medical Education and Hospitals of the American Medical Association[2] at $328,937,777, and the annual maintenance cost for these services at approximately $75,906,582.

Though its incidence is steadily diminishing, tuberculosis is still one of the most pressing problems confronting the Nation because of its seriousness and its preventable character.

The favorable trend in the tuberculosis death rate throughout the United States is, for the most part, to be ascribed on the one hand to improvement in economic status and better housing and dietary standards, and on the other to a diminishing amount of community infection in consequence of education, case-finding and hospitaliza-

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tion. As these factors are to a considerable extent within our control, there is a prospect of the practical elimination of the disease as a serious public-health problem.

REGIONAL VARIATIONS IN THE PROBLEM

The varying magnitude of the problem is apparent when political subdivisions are ranked in accordance with the tuberculosis death rates in 1936. Porto Rico heads the list[3] with a rate of 297.9 and is followed by Arizona (274.6), New Mexico (122.0), District of Columbia (106.1), Tennessee (89.6), Nevada (88.0), Hawaii (85.6), Maryland (85.2), Colorado (76.3) and California (76.2). Without mentioning the states in the intermediate group, an enviable position at the bottom of the list is taken by New Hampshire (33.7), Kansas (28.7), North Dakota (24.9), Idaho (24.7), Iowa (23.8), Utah (21.5), Nebraska (18.3) and finally Wyoming (18.0). The Commonwealth of Massachusetts in 1936 was in thirty-second place among the states in the Union. In 1938, there were 1543 deaths from the pulmonary form of the disease, a rate of 34.9. Deaths from extrapulmonary cases numbered 141, a rate of 3.2. The death rate from all forms in 1938 was therefore 38.1.

The tuberculosis death rate is high in the Negro population, in whom the disease ranks second as a cause of death. The problem is in consequence especially serious in the South.

A high death rate from tuberculosis among Spanish Americans and Mexicans living in the United States is partly responsible for the excessively high rates in some of the counties in Texas, Colorado, New Mexico, Arizona and California.[4]

Migration from one state to another plays a part in the uneven distribution of the disease. Such resort states as Arizona, New Mexico and Colorado are in consequence confronted with an especially difficult problem. Interchange between the states of persons with tuberculosis is to a less extent a problem in all parts of the Union.

Adequate tuberculosis control is in large measure an economic problem, inasmuch as it is concerned with standards of living and the application of measures for diminishing the amount of community infection. Financial resources vary widely among the states. Case-finding facilities and hospitalization of the tuberculous are important factors in control of the disease. A provision of at least two beds per annual death is desirable.

The Technical Committee on Medical Care in its report[5] to the National Health Conference at Washington estimates the ratio of beds per annual death for the United States as a whole at 1.15, with variation for individual states from 2.75 to 0.20. Only five states have two or more beds per annual death, and in twenty-six states the figure is less than one. Nine states make no legal provision for sanatoriums. Five of these subsidize care at local institutions, and four make no statewide provision for the hospitalization of patients.

The South with its large Negro population is under a serious economic handicap and makes little provision for tuberculosis control.

NATION-WIDE TUBERCULOSIS PROGRAM

Tuberculosis control in the United States falls short of an attainable goal. This is due in part to economic factors and in part to lack of understanding, acceptance and application on the part of the public, the medical profession and official agencies of remedial measures. In communities such as Massachusetts, where the facilities are adequate, the problem is largely educational and for its solution demands more widespread support of the program. In many communities, and especially in the South, the situation is more serious on account of the lack of diagnostic facilities and opportunity for adequate institutional care.

It may be assumed that further substantial progress in tuberculosis control will not be accomplished without the inauguration of a uniform and adequate program throughout the country as a whole. Leadership and financial assistance in the solution of the problem should come from the central government at Washington, leaving the actual operation of the project to states and localities.

A nation-wide program for the prevention and control of tuberculosis was formulated by a special committee of the board of directors of the National Tuberculosis Association, and was approved on June 20, 1938.[6]

At the National Health Conference in Washington, July 18-20, 1938, the Interdepartmental Committee presented a national health program. Only that part of the program dealing with tuberculosis need be considered here. The Technical Committee on Medical Care[5] reported in connection with the expansion of general public-health service the need for a concerted attack on certain specific problems of national health, including tuberculosis. It recommended case-finding, especially by x-ray examination of contacts of known cases, isolation and treatment (usually bed care) of persons with active disease and periodic observation of those with latent or quiescent disease.

A draft of a proposed bill imposing additional duties on the United States Public Health Service with respect to tuberculosis control was presented by Mr. Folks at the meeting of the board of directors of the National Tuberculosis Asso-

ciation, February 11, 1939, and was approved in principle as a working basis for federal provision. It is in substance as follows:

The recommendation is made that measures for the prevention and control of tuberculosis be established, extended and improved in the political subdivisions of the United States, especially in those with high death rates from the disease and those suffering from severe economic distress, that studies, investigations and demonstrations be conducted and that personnel be trained to accomplish these purposes.

To carry out this project, it is suggested that Congress authorize the appropriation of funds to be made available and allotted through the United States Public Health Service to political subdivisions on the basis of the population, extent of the problem, existing facilities and financial needs and in accordance with plans presented by the local health authority and approved by the Surgeon General.

It is suggested further that after consultation with the state and territorial health officers, the Surgeon General of the United States Public Health Service be authorized to prescribe the rules and regulations necessary to carry out the plan and that a Division of Tuberculosis Control be established in the United States Public Health Service. The bill suggests for the year ending June 30, 1940, an appropriation of a sum not to exceed $7,750,000; for the year ending June 30, 1941, of a sum not to exceed $33,500,000; for the year ending June 30, 1942, of a sum not to exceed $37,000,000; and for each year thereafter of such sum as may be deemed necessary to carry out the purpose of this act, provided that subsequent to the year 1945 the sum shall not exceed $17,500,000.

The Wagner Bill (Senate 1620) was introduced to be enacted as the National Health Act of 1939, as an amendment to the Social Security Act to carry out some of the purposes of the National Health Program, outlined at the Washington conference. It recommends the provision of more adequate public-health service, prevention and control of disease, maternal and child-health services, construction and maintenance of needed hospitals and health centers, care of the sick, disability insurance and the training of personnel. It advocates the appropriation of certain sums for public-health work and investigation, and grants to states for hospitals and health centers for the control of tuberculosis, among other projects. The provisions of the bill in its wider scope are much the same in principle as suggested by the National Tuberculosis Association with respect to tuberculosis control. In addition, however, the Wagner Bill requires financial participation by the states and provides for federal and local advisory councils.

The sums specified for public-health and medical care in the Wagner Bill are to be allotted to political subdivisions on a matching basis, the highest proportion, two thirds, being applicable to the state with the lowest financial resources, and the lowest, one third, to the state with the highest financial resources. The expenditure of federal funds would amount to nearly $100,000,000 for the first year, without any specific sum's being mentioned for mental disease and tuberculosis hospitals, with gradual increases thereafter. With respect to mental disease and tuberculosis hospitals, there is authorized to be appropriated "a sum sufficient to carry out, in respect to such hospitals, the purposes of this title."

Without attempting to pass judgment on the Wagner Bill or to discuss the relative merits of the different phases of the National Health Program, it is obvious that tuberculosis control is one of the most urgent of them all.

A nation-wide tuberculosis program should include the provision of hospital care for all discovered cases needing such care and case-finding, including x-ray examination of those exposed to the disease by family contact.

NECESSARY HOSPITAL BEDS

Estimate of the number of necessary hospital beds for patients with tuberculosis is based on the desirable ratio of two beds per annual death. In addition to the beds already available, it is estimated by the National Tuberculosis Association that about 40,000 beds are needed for tuberculosis.

With respect to the recommendation of the Technical Committee on Medical Care at the National Health Conference[5] that in a ten-year period hospital facilities should be expanded by the provision of 360,000 beds in general, tuberculosis and mental disease hospitals, Fishbein[7] has made the criticism that hospitals in the United States have been for the past five or six years from 25 to 35 per cent unoccupied. It is also stated regarding general hospitals of the country in 1938 that 31.1 per cent of the beds were unused.[8]

It should be noted, however, with respect to tuberculosis hospitals, which alone are under discussion here, that in a census[2] covering 92,339 beds for tuberculosis patients there were 13,571 vacancies (14.7 per cent) and at the same time a waiting list of 9854 patients. This unequal distribution in patient load may be taken to indicate regional variation in the pressure for beds.

So far as tuberculosis is concerned, it is obvious that the number of available beds has a wide regional variation and is not high enough in the country as a whole. Nevertheless, before embarking on an expensive building program for additional beds for tuberculosis or other purposes it is of course desirable to determine to what extent existing facilities are or can be made available.

In view of the probable continuance of the decline of tuberculosis as a public-health prob-

lem, it is appreciated that the additional beds will not be needed for the project permanently, and that they should be so constructed and located that they can be used later for general or other hospital purposes.

There are nearly three beds per annual death of pulmonary cases in public and private sanatoriums in Massachusetts. The ratio in Massachusetts is exceeded in only few states in the Union and is sufficient for present needs.

CASE-FINDING PROGRAM

In case-finding, the most important method is the x-ray examination of all family contacts of known cases of tuberculosis. The proportion of cases among contacts is larger than it is in any other group. The extent of the case-finding problem in any community may be roughly estimated by multiplying the number of annual deaths from tuberculosis by the number of discoverable cases, using 5 cases per death as the number which can be discovered, and multiplying this result by the estimated number of exposed persons, or 2.4 per family.

Persons reported as dying of tuberculosis, patients in tuberculosis sanatoriums and those with tuberculosis in the practice of physicians are among the groups to which recourse may be had in the case-finding program, and each case so located may serve as the starting point for the investigation of family contacts.

In Massachusetts, tuberculosis, in all its forms, is among the diseases declared dangerous to the public health and reportable through local boards of health to the State Department of Public Health. An exception, however, is made with respect to the childhood type of tuberculosis, concerning which a report from the local board of health to the department is made only when sanatorium treatment is recommended.

It should be noted that, so far as Massachusetts is concerned, the Department of Public Health has recently promulgated rules and regulations which, when carried out, will put this case-finding program into operation. These revised regulations[9] are in part as follows:

> As soon as a diagnosis of tuberculosis has been established, arrangements should be made for the examination, including an x-ray of the chest, of all members of the immediate family and of other persons with whom the patient has been in close contact. If the family cannot afford x-ray examination by a private physician, facilities are available through the various state, county and municipal sanatoriums. Persons with suspicious findings and those who have had contact with a tuberculous patient should be kept under medical observation as long as advised by the physician.

It is fortunate that facilities for this case-finding program are already available in Massachusetts. Since 1931, when the state and county sanatoriums were authorized to extend their services by the maintenance of diagnostic outpatient departments at the request of towns or groups of towns, twenty-two extramural consultation clinics have been established. These clinics furnish diagnostic service through members of the staff of the sanatoriums. They are for the most part in outpatient departments of general hospitals, are provided with x-ray facilities and are available for those who cannot afford to pay.

According to the policies[9] of the Massachusetts Department of Public Health, "It is the responsibility of the local board of health to provide hospital care for cases of tuberculosis when needed, and to see that contacts are examined where such examination cannot be made through a private physician."

With abundant facilities available and plans definitely outlined, there should be an improvement in the case-finding program in Massachusetts. A large proportion of the family contacts of patients in the state and county sanatoriums are now examined by x-ray, but the percentage should be increased in some parts of the State.

In some of the municipal sanatoriums, x-ray examination is likewise made of a considerable proportion of the family contacts. But there is need for improvement in these institutions in the extension of the investigation to include all family contacts. Lack of adequate facilities is in part responsible for the incompleteness of the case-finding program. This difficulty can be surmounted by making use of the services of the state and county sanatoriums.

Case-finding in school children has been in operation since 1924. The school program suffers from failure to secure parental consent for the investigation of more than 50 to 60 per cent of the children. The advantages of the finding of tuberculosis in school children are twofold — to the affected child and to the community. Yet these advantages are fully realized only when the investigation includes both children and family contacts. In general, little has thus far been done to round out this part of the program and to examine by x-ray the family contacts of the children with the childhood (hilus) as well as the adult type of tuberculosis.

The examination of a larger proportion of the family contacts of tuberculous patients in the practice of physicians may be promoted by local boards of health through a circular letter to physicians asking for a list of all tuberculous patients under their care during the year, whether or not previously

reported, emphasizing the importance of sputum examination in suspicious cases, calling attention to the availability of the State Bacteriological Laboratory or other approved laboratories, noting the importance of the x-ray in the early diagnosis of the disease and listing the facilities in the State for the x-ray examination of patients and contacts unable to pay.

Group investigation in Massachusetts should be extended to include all teachers, medical students, hospital interns and nurses, college students, diabetic patients, and nursemaids and domestic help in homes where there are children.

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REPORT ON MEDICAL PROGRESS

PATHOLOGY

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BOSTON

TO REVIEW, in a few pages, progress in pathology, the least specialized and most inclusive division of medicine, is a manifest impossibility. Probably the editor consciously or subconsciously realized this in assigning to a score of reviewers in this series so many of the choicest items on the pathologic bill of fare that little is left to the pathologist save morbid anatomy in the strictest sense and experimental work in fields still devoid of practical clinical application. Even so, the field remains enormous and a purely arbitrary selection must be made. Several subjects stand out as worthy of brief review because the work of several investigators with varying viewpoints and technics has focused closely enough to permit comparison. Progress is not always easy to distinguish from regress, and in the assessment of rival claims it seems most honest to make no attempt to conceal one's inevitable bias.

ARTERIOSCLEROSIS

In the last five years there has been in this country a recrudescence of lively interest in the subject of arteriosclerosis. In a series of papers documented by numerous photomicrographs of exceptional clarity, Leary[1-3] has compared in detail human lesions with the experimental ones so readily produced in rabbits by the feeding of diets rich in cholesterol. In the rabbit it can be shown that in the process of cholesterol deposit fibroblasts are stimulated to multiply and to lay down collagen. With withdrawal of the cholesterol from the diet the lipid gradually disappears from the vessel but the fibrous intimal thickening persists. By such methods every phase of the human disease can be accurately mimicked. Leary feels that he has demonstrated so close a similarity in the lesions in the two species that one is justified in considering them identical in pathogenesis, and on this ground he advances the hypothesis that human arteriosclerosis is dependent on an abnormality of the cholesterol metabolism. He admits, but does not stress, the importance of local vascular phenomena in determining the localization of the deposits. In subsequent communications he has somewhat qualified this conclusion, admitting that the disorder in cholesterol metabolism may be local rather than general.

Duff,[4,5] in contrast, finds slight but undeniable differences between the rabbit and human lesions, particularly in an early involvement of the media in the experimental lesions which is lacking in the human disease. He presents evidence of preliminary degenerative change in the arterial walls before cholesterol is deposited, and believes that hypercholesterolemia alone will not give rise to atheroma. He emphasizes the lack of clinical or extravascular histological evidence of disordered cholesterol metabolism in the human disease except in relation to diabetes, where the disturbance of metabolism is so extensive that many other factors besides the hypercholesterolemia may be responsible for the initiation of the lesion. He calls attention once again to the well-known fact that premature arteriosclerosis has never been experimentally produced by cholesterol feeding or in any other way in omnivorous animals which spontane-

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ously develop the disease at advanced ages, and wisely advises caution in applying to man a conclusion which seems self-evident from a study of the artificial rabbit disease.

Experimental rabbit atherosclerosis remains, however, a fascinating tool for the investigative pathologist. Systematically the variations are being played; testing, for instance, one after another endocrine gland to determine whether over- or underfunction will accelerate or decrease the rate of development of vascular lesions. To date the most consistent results relate to the thyroid,[6] where hyperthyroidism is generally agreed to delay the appearance of atheroma and hypothyroidism to accelerate and accentuate it — a point not to be forgotten before recommending total thyroidectomy for the relief of angina pectoris. Such substances as thyrotropic pituitary hormone[7] and potassium iodide,[6, 8] the effects of which on experimental atheroma are notably variable, can fairly safely be assumed to act through the mediation of altered thyroid function. Physicians with the requisite philosophy may find complacence in the experiments of Eberhard,[9] who demonstrated that though alcohol ingested along with cholesterol raised the level of blood cholesterol, it delayed the deposit of the lipid in the blood vessels.

A new angle of attack on the problem has been developed and energetically pursued under the direction of Winternitz at Yale University. With various collaborators he has made an extensive study of the vasa vasorum of the aorta and various major vessels, such as the coronary and renal arteries, under normal and pathologic conditions. The results of this work have been summarized in a monograph rather broadly entitled *The Biology of Arteriosclerosis*[10] replete with colored illustrations which support the author's points with convincing clarity and esthetic charm. Conservative in positive statement, it is presented in a manner which permits almost limitless implications. Although vascularization of atherosclerotic plaques has been repeatedly noted by previous investigators, its extent has certainly never been so vividly demonstrated. A critical study of both the illustrations and the text, however, leaves little doubt that the great majority of lesions studied must be classified as advanced and as derived from vessels with long-standing disease. Convincing evidence that in normal human arteries the vasa vasorum extend into the intima, or even with significant frequency into the inner third of the media, is lacking. Under such conditions it is difficult to believe that lesions of the vasa vasorum can initiate atherosclerotic plaques, which all evidence indicates are, primarily, intimal lesions. The subintimal hemorrhages which the author finds with such frequency by his method of clearing vessels with the Spaltheholz method are of interest and have undoubtedly been neglected by most previous investigators. Though hemorrhages may evidently be invisible beneath a thickened opaque intima, it seems scarcely possible that they could be missed by any method of examination in a vessel with an intima of normal thickness which is almost perfectly transparent. It seems doubtful, therefore, that such hemorrhages can, as Winternitz suggests, be a significant factor in the initiation of atherosclerotic lesions. That they may, however, play a vital role in the disruption of established atheromatous plaques and in the initiation of thrombosis seems more probable. This possibility has been emphasized by Paterson,[11, 12] who found intimal hemorrhages arising, he believes, from the vasa vasorum in 32 of 37 consecutive cases of coronary thrombosis. Continued extensive investigation by a variety of methods will be necessary to establish the validity of this hypothesis as against Leary's theory of the anemic necrosis of the thickened avascular intima. The application of Winternitz's technic to the study of experimental atheroma might go far in answering some of these questions. On all investigators working with human material the importance of establishing adequate criteria for determining the age of the lesions under observation, and the particular importance of studying early lesions should be urged.

CORONARY SCLEROSIS AND INFARCTION OF THE HEART

The correlation of infarction of the myocardium with occlusion of the coronary artery is often difficult or even impossible under the conditions of a routine autopsy. All pathologists frequently see thromboses unassociated with infarction, and acute infarcts associated with a vessel so completely fibrosed and calcified that it must have been occluded years before. The existence of collateral circulation has, of course, been inferred in order to get them out of their difficulty. More precise information in regard to this collateral has been accumulating rapidly in recent years. By extreme care and thoroughness in the gross examination of the coronary circulation, Saphir and his collaborators[13] were able to show that infarction of the myocardium rarely develops when a single coronary branch is occluded unless there is simultaneous or pre-existing partial or complete obliteration of other branches as well. An acute infarct, for instance, may be found in the area normally supplied by the descending branch of the left coronary; this vessel will show an old occlusion, but with sufficient care a fresh occlusion can always be found

elsewhere, perhaps in one of the branches of the right coronary artery which has for years been supplying a collateral circulation. Though collateral circulation may safely be inferred from such observations, it cannot ordinarily be demonstrated by simple methods of gross dissection. A notable advance, therefore, is represented by the recent work of Schlesinger.[14] He uses a new injection mass of agar and lead salts which permits a very uniform injection of the arterial tree down to arterioles of 40 microns in diameter. He has in addition developed a new technic for opening, or rather unrolling, the heart which without cutting across any important vessels enables one to spread out the entire heart in one plane. This enormously improves the radiographic visualization. With this method Schlesinger has found in truly normal hearts no anastomoses large enough to permit the passage of his injection mass. In diseased hearts of various types they are frequently found, and in cases of coronary sclerosis they can be readily demonstrated whenever arteriosclerotic narrowing or occlusion causes obstruction in the coronary circulation. This extensive spontaneous development of collateral circulation should be borne in mind in interpreting the results of operations designed to produce artificially such a collateral circulation.

LUNGS

A point of minute anatomy in the lungs which is of great theoretical importance to the pathologist seems finally to have been settled after years of dispute with the histologist. This concerns the existence of pores of Cohn in normal lungs. Macklin,[15] by a study of very thick lung sections in which many alveolar walls were viewed from their flattened surfaces rather than in ordinary sections, was able to show obvious communications between alveoli in various animals and in man. Though present at all ages, they become larger and more numerous with advancing age. Loosli[16] has confirmed this finding by the method of serial sections, in an article documented by photomicrography of exceptional clarity and detail. Their minute size, 5 or 10 microns, explains why they were missed in previous reconstructions. The importance of such interalveolar air channels in explaining the relation of bronchial plugging to atelectasis and the mode of spread of infection throughout a lobe are obvious.

In the field of pulmonary neoplasm, more and more attention is being given by bronchoscopists, thoracic surgeons and pathologists to those peculiar semi-benign bronchial tumors which have generally in this country been called adenomas. It is evident that in some clinics they are even yet not distinguished from carcinoma. Though often locally infiltrative and unquestionably capable of metastasis to regional nodes, their progress is so slow and the likelihood of generalized metastasis so slight that they deserve to be sharply distinguished. Attempts to explain their histogenesis have been numerous and unsatisfactory. Womack and Graham[17] have recently proposed that they are mixed tumors and should be included with the hamartomas. Hamperl[18] in contrast points out the remarkable histological and clinical similarity to the argentaffine tumors of the intestinal tract ordinarily called carcinoids. Decision upon such a point of pathogenesis must rest primarily on accurate histologic observation, the first prerequisite of which is good histologic technic. A glance at the photomicrographs accompanying the two articles would suffice to explain why the reviewer's prejudices lie with Hamperl despite the uniform failure of all investigators to demonstrate that the cells are argentaffine.

The development by Robertson and his collaborators[19–22] a few years ago of a reliable method of producing pneumococcal lobar pneumonia in dogs has added much to our knowledge of the pathologic lesions of pneumonia. The study of a very large number of experimental lesions and careful comparison with human material have made it apparent that lobar pneumonia ordinarily starts as a focus of parenchymal infection near the periphery of a lobe. Spread occurs via the air passages and the pores of Cohn, and Blake and Cecil's[23] theory of lymphatic extension has not been supported. Loeschcke[24] from a very careful study of extensive human material had already reached the same conclusions, and his findings are in close agreement with those of Robertson.

In a continuation of these studies Robertson and his associates[25, 26] have shown clearly that recovery from pneumonia depends on a dual mechanism: first, a generalized process which acts to localize the infection and prevent or control invasion of the blood stream; and second, a local process by which the lesion itself is finally freed from microorganisms. To a large extent these processes appear to be independent of each other. The generalized element is the familiar process of antibody formation; the local one is a tissue reaction within the lung itself. The outstanding feature of the latter is a macrophage reaction which appears about the fortieth hour even in animals which may eventually succumb to bacteremia. With the onset of this macrophage reaction the lungs very rapidly become sterile and pneumococci, when found at all, are almost entirely intracellular. It is possible for such a macrophage reaction to sterilize one lobe while active progression of in-

fection is occurring in other lobes. The persistence and even occasional extension of consolidation following apparent symptomatic cure by serotherapy are explained by this independence of the two reactions. They will soon have to be carefully evaluated in relation to chemotherapy.

TUBERCULOSIS

In no field of pathology is progress more difficult to assess than in tuberculosis, particularly its experimental aspects. Hardly an experiment can be recorded for which in the ensuing years refutations do not outnumber the confirmations. Uncontrolled — perhaps uncontrollable — variations in dosage, virulence, natural host resistance, time of observation, dietary factors and complications of intercurrent disease render the same experiment in two investigators' hands barely comparable. It may be of value to point out some of these conflicting results and so drive home the need of deliberation in the acceptance of any experimental results in this field.

One of the sources of liveliest debate has been the relation of allergy to immunity. Rich[27, 28] and Cannon and Hartley[29] among others, have shown clearly that allergic inflammation fails to localize a virulent pyogenic organism such as the pneumococcus. This of course does not prove that it may not localize tubercle bacilli. Apparently conclusive evidence that also in tuberculosis immunity can be separated from allergy was presented by Rothschild, Friedenwald and Bernstein[30] when they showed that infected animals desensitized with tuberculin showed less extensive tuberculosis two months after re-infection than did allergic animals or normal controls. Although this conclusion was confirmed by Birkhaug[31] and various other investigators,[32] results pointing in the opposite direction were obtained by Willis and his collaborators.[33, 34] In an extensive experiment with over 500 animals they found that when the observations were carried beyond the sixty-day period used by Rothschild et al. the desensitized animals, although developing fewer tubercles in the liver and the spleen, died in large numbers of acute tuberculous pneumonia long before the allergic control animals. In all such experiments the repeated injections of large amounts of tuberculin necessary to keep the animals desensitized provoke fever, anorexia, loss of weight and the death of numerous animals either from shock or intercurrent disease, and the results are almost impossible to interpret. Moreover, the failure to evoke a skin reaction in such debilitated animals is by no means conclusive proof of complete desensitization.

Another subject which continues to be the source of a voluminous literature is the study of the factors underlying the formation of the tubercle and their relation to allergy. Under the aegis of the National Tuberculosis Association an extensive collaborative enterprise involving many laboratories and scores of workers has been centered about the chemical fractionation of the tubercle bacillus and the testing of the various separate and combined fractions histologically and immunologically. Many of the early results proved misleading or contradictory — first, because many of the earlier fractions tested were impure; secondly, because the significance of the degree of allergy was not always borne in mind; thirdly, because quantitative probabilities were not duly considered; and finally, because in the process of purification proteins have probably been chemically and antigenically altered. In Sabin's most recent publications[35–39] many of these conflicts appear to have been resolved. The carbohydrate fraction will not induce sensitivity and gives only an immediate polymorphonuclear type of skin reaction; the waxes provoke only nonspecific foreign-body reactions; the proteins will induce sensitivity and produce either polymorphonuclear or mononuclear response, dependent, as Dienes and Mallory[40, 41] pointed out for proteins in general, on dosage and degree of allergy; the phospholipids produce an accentuated mononuclear response with marked development of epithelioid cells. By combining phospholipid and protein, apparent true tubercle formation is induced and hypersensitivity greatly increased. The amount of phospholipid employed is still far greater than the dissolution of a reasonable number of tubercle bacilli would provide, and the reaction may not be entirely specific. An accentuation of hypersensitivity by the combination of tubercle bacilli or their derivatives with other fats and oils has been noted by Saenz[42] and others. Still more conducive to conservatism in attributing to specific organic chemical factors of the tubercle bacillus responsibility for the development of one or another feature of the exudative reaction is the demonstration by Gardner[43] that every phase of this reaction can be exactly duplicated by the injection of a simple inorganic substance, silicon dioxide.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 25251

PRESENTATION OF CASE

An eighty-three-year-old man was admitted complaining of chills and fever.

Six days before entry the patient noted the onset of a chilly sensation. His head felt big, and he had anorexia. He went to bed early, slept well, but woke the following morning and noticed increased perspiration. He had no appetite and remained in bed most of the day. His nose felt stuffed up, and his head full. Postnasal phlegm, which was worse when he had a cold, had been a chronic complaint for the past several years. His physician saw him on this second day of his illness and found a temperature of 98.6°F. and a pulse of 72. There were a few rales at the bases of both lungs. The following day he had a chill followed by a temperature of 103°F. and respirations of 30. Physical signs on examination of the lungs were indefinite. There was no pleural pain. The blood showed a white-cell count of 15,750 with 94 per cent polymorphonuclears. The blood pressure was 128 systolic, 60 diastolic. It was assumed that he had pneumonia, and 9.5 gm. of sulfapyridine were given at four-hour intervals during the following twenty-four hours. Shortly after completion of this course of therapy he had another chill and his temperature rose to 101.6°F. A few hours later he was again given 2 gm. of sulfapyridine, following which his temperature soon fell to 99.2°F. On examination the tongue and soft palate were red. The lungs showed no definite signs except for slight dullness at the right base and a few rales at the left base. Respirations were 30. On the following day he seemed better during the morning but in the afternoon again had a chill with a temperature rise to 101°F. An x-ray film of the chest, taken with a portable machine, showed no evidence of pneumonia. He had not had abdominal pain or tenderness, jaundice or urinary symptoms.

Physical examination on admission showed a well-developed and nourished, acutely ill man. There were a few rales at both lung bases. The blood pressure was 128 systolic, 60 diastolic. No other definite physical signs were noted.

The temperature was 100°F., the pulse 100, and the respirations 27.

Examination of the urine showed a specific gravity of 1.020, a trace of albumin, a slight trace of bile, many coarsely granular casts, rare cellular casts, 3 or 4 white cells and an occasional red cell per high-power field, no bacteria and no sugar. The blood showed a red-cell count of 4,520,000 with 90 per cent hemoglobin, and a white-cell count of 27,400 with 83 per cent polymorphonuclears, 4 per cent small lymphocytes, 11 per cent mononuclears, 1 per cent nucleated red cells and 1 per cent myelocytes. The red cells appeared fairly normal; there were rare polychromatic and stippled cells. The nonprotein nitrogen of the blood serum was 65 mg. per 100 cc. Blood cultures showed *Bacillus mucosus capsulatus* (Friedländer's bacillus), as did the urine cultures.

Portable x-ray films of the chest showed a band of linear density extending upward and laterally from the left hilus to the lateral chest wall in the region of the interlobar pleura. There were oval areas of rarefaction just beneath the pleural surface in this area. The lung markings extending to the right base were a little prominent, and there was some haziness just above the midportion of the right diaphragm. There was a large calcified node in the right hilus. The diaphragm was high in position. The costophrenic angles were clear. The aorta was tortuous, and there was calcification in the arch.

The patient had another chill on the afternoon of admission. Sulfanilamide therapy was begun. On the following day his temperature was 103.8°F. There were many rales at the right base posteriorly. On the third hospital day the patient was drowsy and could not be aroused. The right lung was clear, but rales were heard at the left base. The abdomen was negative. There was slight stiffness of the neck. The white-cell count was 32,700 with 96 per cent polymorphonuclears. The temperature was 101 to 103°F. On the following day his neck was stiff and rigid. The left chest was clear, but rales were present on the right. The nonprotein nitrogen of the blood serum was 95 mg. per 100 cc. On the fifth hospital day he still could not be roused. His pulse was very weak. There was slight edema of the ankles. The abdomen was slightly distended. There was slight jaundice. His neck remained stiff. He rapidly failed and died on the sixth hospital day.

DIFFERENTIAL DIAGNOSIS

DR. CHARLES L. SHORT: May we see the x-rays?

DR. AUBREY O. HAMPTON: These portable films were taken in this hospital. The linear shadows described in the left lung follow the course of the pleura between the upper and lower lobes. You sometimes see them following pleurisy; they are

also associated with tuberculosis. These oval areas of rarefaction which simulate thin-walled chronic cavities are just as likely to be thickened pleural septa with areas of emphysematous lung between.

DR. SHORT: This represents an old process?

DR. HAMPTON: Yes. I think that everything we see in the chest is chronic. This shadow at the right base could be acute consolidation but it is seen in only one film and it is not plain. I do not see how we could make a diagnosis of active disease in his chest.

DR. SHORT: To save time I have decided to admit from the beginning that this patient had an infection with *Bacillus mucosus capsulatus* or Friedländer's bacillus. I can hardly believe that both the blood culture and urine culture represent contaminants, nor, since the blood culture was taken six days before the patient's death, do I think that the former could represent an antemortem invasion of the blood stream by essentially saprophytic organisms. Of course he may have had a combined infection; the picture is suggestive of acute miliary tuberculosis, with indefinite signs and lack of a definite x-ray picture in the lungs, and terminal meningitis. As Dr. Hampton said, there is possibly some evidence of an old tuberculous process in the chest. I do not believe we have enough positive findings to make that diagnosis and, therefore, we have to dismiss it. I do not see that any of the picture represents toxic symptoms from sulfapyridine, although he was given a large dose in the first twenty-four hours. There is no evidence of agranulocytosis or diminished resistance to infection and, surprisingly enough, no mention was made of his vomiting.

The points left for us to decide, then, are the primary source of the bacteremia and the organs of the body which were finally invaded. I think that from the negative x-ray of the chest and the lack of definite signs on physical examination we can rule out a primary pneumonia. The lung findings may represent either an old process, as Dr. Hampton suggests, or possibly an early metastatic infection in the lungs. According to the literature, extrapulmonary infections with this organism are not uncommon. Baehr, Shwartzman and Greenspan* reported 198 cases from one hospital. Of these, nearly a third resulted from peritonitis following a perforation of the gastrointestinal tract. The next commonest source was the biliary tract or the liver, and the next the genitourinary tract. In these last two, stasis was a factor and there was often a bacteremia. There were very few cases originating in the middle ear or sinuses.

*Baehr, G.; Shwartzman, G., and Greenspan, E. B.: Bacillus Friedländer infections. Ann. Int. Med. 10:1788-1801, 1937.

The onset of this patient's illness was certainly not very characteristic. It suggested a head cold until the development of chills and fever. I think a presumptive diagnosis of pneumonia was probably justified from the clinical picture in a man of his age. He apparently had jaundice on admission because of the finding of bile in the urine, although it was not noted in his skin or sclerae until a day or two before he died. I do not believe there is enough evidence for a primary infection in the liver or biliary tract. The jaundice is really the only thing we have pointing in that direction and it could just as well be a toxic hepatitis due to a generalized infection.

Then we come to the genitourinary tract. The patient died with a high nonprotein nitrogen in the blood serum. We are not told anything about the fluid intake or how much he vomited. Probably he did not take in very much in the last few days when he was in coma. The one examination of the urine hardly suggests a urinary-tract infection, but it does not rule it out. Of course, with a generalized infection, the positive urine cultures could be explained on the basis of excretion through the kidneys of the organisms in the blood stream. There is nothing to suggest that this patient had an already existing nephritis. He had a normal blood pressure and fairly good specific gravity of the urine. I shall have to assume that, unless the high nonprotein nitrogen was due to vomiting and dehydration, he did have a renal infection which was severe enough to destroy most of the functioning tissue in his kidneys. This may have been furthered by some unrecognized urinary obstruction, perhaps an enlarged prostate.

Terminally the patient had signs of meningitis. Of course, without lumbar puncture we have no way of knowing that he did not have an extradural abscess which may have given these signs with a sterile spinal fluid. To sum up, I think this patient had a generalized Friedländer's infection with bacteremia, with the primary source undetermined. We cannot say he had a primary pneumonia or primary infection in the biliary tract or liver. There is no real evidence of sinus or middle-ear infection. By elimination, a primary source in the urinary tract is most likely, resulting in a severe pyelonephritis, and finally the development of signs of meningitis.

CLINICAL DIAGNOSES

Friedländer's bacillus pneumonia.
Thrombosis of basilar artery?

DR. SHORT'S DIAGNOSES

Bacteremia, Friedländer's bacillus.
Pyelonephritis.
Meningitis.

Anatomical Diagnoses

Septicemia, *Bacillus mucosus capsulatus*.
Multiple abscesses of the liver.
Healed pulmonary tuberculosis.

Pathological Discussion

Dr. Tracy B. Mallory: Dr. Short has done as well as we could at the autopsy. We could not find the source of infection. We did not have permission to do the head, so one of the accessory sinuses may possibly have provided it. We did do the spinal cord, however, and he had no diffuse meningitis. The most obvious findings at autopsy were multiple abscesses in the liver, the right lobe being almost completely filled with them. It is often difficult with extensive liver involvement to determine the mechanism of infection. Nothing was found, however, to suggest cholangitis or pylephlebitis, so that I believe the organisms reached the liver via the hepatic artery as part of the general septicemia. The lungs were entirely negative, except for extensive healed tuberculosis. The kidneys showed vascular nephritis and the ordinary cloudy swelling due, I am sure, to his fever and toxemia. There was no pyelonephritis, and though the prostate was a little large, it was not hard and did not appear to be obstructing, so I do not think the urinary tract was the source of infection.

CASE 25252

Presentation of Case

First Admission. A thirty-eight-year-old widow was admitted complaining of dysuria.

She had noticed burning at the end of micturition of seven days' duration. Frequency and urgency were also noted. Gram-negative intracellular diplococci were found. Diagnoses of cystitis and acute urethritis were made. She was discharged on the eighteenth hospital day.

Second Admission (seven years later). Her menses had been normal and regular until four months before admission when she had five attacks of intermenstrual bleeding during one month. Each was somewhat less in amount than that of a normal period. Following this she had daily spotting until three weeks before entry when a diagnosis of carcinoma of the cervix, confirmed by biopsy, was made at another hospital. She received 4500 r of x-ray therapy during the next twenty-one days. After the treatment was begun the discharge changed from bloody to serous. She had continued to have periods at the regular time, the last one twenty-six days before entry.

Physical examination showed a well-developed and nourished woman in no distress. Examination of the chest was negative except for an occasional rale at the right apex. The blood pressure was 120 systolic, 80 diastolic. An apparent x-ray burn was present on the skin of the lower abdomen. Under nitrous oxide anesthesia, pelvic examination showed a 9-cm. retroverted uterus which could be replaced. There was apparent submucous extension of the cancer at the external os, but the lesion seemed fairly superficial; a biopsy was taken. Radium needles were implanted, the total dose being 3000 hours. The pathological report on the biopsy was acute and chronic inflammation.

On the fourth hospital day the patient vomited several times. The abdomen was soft. No further vomiting occurred, however. Her chart remained flat, and she was discharged on the tenth hospital day.

Third Admission (seven months later). For several months after discharge the patient had weak spells almost every day but gained a small amount of weight. She had no vaginal bleeding and no further menses. One week before admission she noted the onset of rapidly increasing weakness followed three days later by a severe mid-abdominal pain located for the most part about the umbilicus and radiating through to the back. At the same time, nausea, vomiting and constipation developed. Her abdomen then became distended. On the afternoon of the day of admission an enema was given, with expulsion of a large amount of black fecal material. Following this her pain was greatly relieved, but recurred and was relieved by 1 gr. of codeine.

Physical examination showed a well-developed and nourished woman in no acute distress. The skin of the lower mid-abdomen showed increased pigmentation and induration over a 15-cm. area. Examination of the chest was negative. The blood pressure was 118 systolic, 80 diastolic. The abdomen was soft, with active peristalsis. There was acute right lower-quadrant tenderness, but no spasm. There was also rebound tenderness referred to the midline.

The temperature was 98.6°F., the pulse 80, and the respirations 17.

The urine examination was negative. The blood showed a red-cell count of 4,410,000 with 80 per cent hemoglobin, and a white-cell count of 10,900 with 87 per cent polymorphonuclears. The smear showed several band forms of polymorphonuclears, a young polymorphonuclear and an atypical mononuclear. Some of the red cells appeared somewhat larger than normal. The serum protein was 5.5 gm. per 100 cc. Two stool examinations

were guaiac positive. The sedimentation rate was 3 mm. in fifteen minutes, 7 mm. in thirty minutes, 12 mm. in forty-five minutes and 15 mm. in sixty minutes.

X-ray films showed a soft-tissue mass about 4 cm. in diameter in the left side of the true pelvis. There was fecal material in the bowel, and the right kidney area showed multiple areas of density. An intravenous pyelogram was negative. The lumbar vertebrae showed lateral curvature, apparently due to anatomical variation of the fifth lumbar vertebra and upper segment of the sacrum. There was no evidence of metastatic malignancy in the bone. On the following day, x-ray films showed no change in the soft-tissue thickening in the pelvis. There was more gas in the small bowel on the left side of the abdomen. The loops were definitely dilated, and one extended to the true pelvis. There also was gas in the colon. Although the gas in the small bowel indicated some obstruction, it apparently was not complete.

She did not improve, and on the fourth hospital day an operation was performed.

Differential Diagnosis

Dr. Oliver Cope: I should like to narrow down the diagnosis, if I can, before Dr. Hampton shows us the x-ray films.

A brief review of the history shows a first episode of acute gonorrhea. Seven years later there was a period of abnormal bleeding; a diagnosis was made of carcinoma of the cervix but we are not told anything definite about it. She had x-ray treatment, then radium, with complete relief of the abnormal bleeding and cessation of the menses. In the first month after the irradiation there appeared these transient periods of weakness, occurring frequently, which may have been menopausal symptoms or may have been associated with what appears later. Before she entered the hospital she had a more profound spell of weakness accompanied by pain, and subsequently we find that there must undoubtedly have been an intestinal hemorrhage and also some bowel obstruction. X-ray study gives further evidence of a mass in the left side of the pelvis and of small-bowel obstruction, which is consistent with the history.

The diagnosis has to take in two important things: small-bowel obstruction and intestinal hemorrhage. The physical findings are of importance. At the time of the second admission, when the radium was given under an anesthetic, no mass was felt in the pelvis. The mass shown by x-ray is, therefore, something that has arisen in the past seven months. I shall exclude certain diagnoses very rapidly. In the first place, inflammatory lesions such as appendicitis and diverticulitis should be excluded on the basis of intestinal hemorrhage. Endometriosis, which occasionally might cause intestinal hemorrhage and may also give small-bowel obstruction, I exclude on the basis of the artificial menopause which had been produced seven or eight months previously. There ought to have been no recurrence of endometriosis, had it been present before the irradiation. I also exclude simple intestinal obstruction in view of the intestinal hemorrhage. In the same way I exclude pelvic inflammation with necrosis following irradiation; I have not seen intestinal hemorrhage under these circumstances. That narrows the diagnosis down to two possibilities: metastatic malignancy from the cervix to the bowel and Meckel's diverticulum or a primary malignant tumor in the small intestine. When we consider metastatic malignancy, I think we can narrow the field still further. I have never seen an ordinary carcinoma of the cervix give rise to intestinal hemorrhage and to small-bowel obstruction — that may be due to lack of experience; an adenoacanthoma, however, does. I have seen one case in which an adenoacanthoma had a secondary lesion in the large intestine with both bleeding and obstruction. I think, therefore, an adenoacanthoma is the most logical way of tying up carcinoma of the uterus and the present symptoms.

If it were not for the history of a lesion in the uterus I should say that the history for which the patient entered the hospital would be best explained by a Meckel's diverticulum. Such a diverticulum characteristically gives bouts of hemorrhage and when acutely inflamed leads to intestinal obstruction. I shall leave it with these two diagnoses, adenoacanthoma and Meckel's diverticulum, until we hear what Dr. Hampton has to say.

Dr. Aubrey O. Hampton: This patient had an x-ray of the spine and chest for metastases, and none were found. This film was taken on the first day, when she had very little evidence of intestinal obstruction. This was the shadow described on the left side, which was thought to be a round mass. It is not so sharp and distinct as a tumor would be. It could be a loop of dilated bowel just as well. Usually the outline of a cyst or tumor is seen all the way around, but this shadow is only partially outlined. That may be because it is tubular; it could not be circular. This soft-tissue shadow is better seen in the film taken at the time of the intravenous pyelogram examination. We can see the bladder and then a perfectly smooth mass that looks like the uterus sitting on top of it. At this examination two days later we see the small bowel dilated down to the region of the true pelvis. That is evidence enough of dis-

ease in or around the small bowel. We have fair or definite evidence that the small bowel is partially obstructed and that it was an intermittent obstruction.

Dr. Cope: It looks as if we were dealing with a true tumor, and the apparent tumor is obstructed bowel. It is very difficult to say on the evidence what the type of cancer may have been, if indeed malignancy was present. The record hedges on this point, saying that the diagnosis of cancer was made at another hospital. It is perfectly possible that the bleeding may have been due to ovarian dysfunction with abnormal endometrium and not to cancer of the cervix or endometrium. An ovarian dysfunction, however, would not explain the intestinal obstruction unless it were one of those things that we hear about — radiation reaction in the presence of an old pelvic inflammation, with secondary obstruction. That does not account for the intestinal hemorrhage. The hemorrhage suggests either that a cancer is present or that it comes from an ulcer in a Meckel's diverticulum. In the absence of definite evidence, the most likely diagnosis is cancer in or around the uterus, with secondary involvement of the small bowel, obstruction and hemorrhage. Adenoacanthoma is the one thing in my experience that would fit the picture and that is my first diagnosis. My second diagnosis is Meckel's diverticulum. I believe it is possible that the original hemorrhages were on the basis of an ovarian dysfunction due to chronic pelvic inflammation, that the diagnosis of cancer was an error and that the heavy irradiation led to intestinal obstruction.

Dr. Tracy B. Mallory: It is obviously a difficult diagnostic problem. Has anyone else any suggestions to offer?

Dr. Horatio Rogers: I should think that x-ray necrosis of the intestine would have to be considered more seriously.

Dr. Cope: Did you ever see hemorrhage following it?

Dr. Hampton: And does not the hemorrhage usually occur at the time of maximum x-ray reaction?

Dr. Rogers: Dr. Frank Pemberton has had some cases at the Free Hospital in which the hemorrhage came on quite late, several months after treatment.

Dr. Hampton: I treated this woman and I gave her most of the treatment in front, crossfiring the midline. She took it very easily without nausea, and I could give her 300 r daily. She was given about 3500 r in front and the other 1000 r behind, with no very obvious reaction. We have given that same dose routinely without any such thing as this occurring and without risk of radiation necrosis.

Clinical Diagnosis

Intestinal obstruction.

Dr. Cope's Diagnosis

Adenoacanthoma of uterus and small intestine.

Anatomical Diagnosis

Post-radiation enteritis.

Pathological Discussion

Dr. Mallory: I shall read you part of Dr. Langdon Parsons's operative notes since he is not here to tell us about the findings:

> On entering the peritoneal cavity, there was an escape of a quantity of thin, peritoneal fluid — obviously in response to an inflammatory condition within the abdomen: this was soon identified as being present in the distal portion of the ileum about 45 cm. from the ileocecal valve and represented a large, pie-shaped area involving the root of the mesentery which was acutely edematous and thickened, about 2.5 cm. in depth. The bowel itself was involved for a distance of about 45 cm. and was loosely adherent to the top of the fundus; this could be easily stripped off with the examining finger without denuding the bowel. It had obviously not been involved in the radiation from below. While the uterus was fixed, there seemed to be no evidence of pre-existing carcinoma of the cervix and the fixation was probably due to fibrosis. The bowel in the involved area showed spots suggestive of gangrene. The entire area was devascularized; the walls were markedly thickened and contracted. The lumen was from 2 to 3 cm. in diameter. There was no marked evidence of dilatation above the point of obstruction. The entire appearance was that of an area exposed to heavy radiation. There was no suggestion of perforation of the bowel. This large loop was freed with great difficulty; the difficulty came in that the bowel was so stiff and edematous that it did not appear in the wound as normal small bowel would, even after freeing it from the uterus, but tended to bend, much as a lead pipe would. The balance of the mesentery appeared normal.

It was very obvious when the specimen reached the laboratory that the bowel wall was of unusual stiffness, and I can well believe that the operator faced a great many difficulties in manipulating it. On opening it we found hemorrhagic necrosis of the mucosa, with extension down through all layers of the bowel wall. Microscopically the most striking feature, perhaps, was the presence in many parts of the specimen, but particularly I should say in the mesentery, of fibroblasts of most unusual character — four or five times the normal size with giant nuclei and giant nucleoli and very frequently five or six nuclei in a single fibroblast, the type of cell one ordinarily considers evidence of a high grade of malignancy. The one common

source for such cells, except within malignant tumors, is in tissue that has been heavily radiated. I consider that finding very characteristic of post-radiation effect, and I think there is no doubt that that was the cause of the localized lesion. Certainly the picture was quite unlike that of regional ileitis, the only other condition we must seriously consider.

DR. HAMPTON: In retrospect we believe that the loop of diseased small bowel was adherent to the fundus and hence lay in the same place throughout the treatment so that by crossfire treatment the maximum dose was delivered to it.

DR. MALLORY: Under ordinary circumstances is it fair to assume that during such a series of treatments several loops would be in your field from time to time rather than one?

DR. HAMPTON: Yes.

DR. COPE: What was the lesion for which she was given the radiation?

DR. MALLORY: Carcinoma. There was never any doubt about that. So far as can be made out at the present time it has been cured.

A PHYSICIAN: Do you think the episode of vomiting represented an acute reaction in the loop of bowel?

DR. MALLORY: I do not know; it may have been that or x-ray sickness.

DR. HAMPTON: She took radiation well and worked throughout the course of treatment; there was no roentgen sickness.

A PHYSICIAN: It says she vomited all day. Presumably after the radium needles were inserted?

DR. HAMPTON: Well, perhaps so.

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THE DEFINITIVE BIOPSY

A recent number of the *Cancer Bulletin*,[1] has commented on Ewing's[2] ideas concerning the use and abuse of the diagnostic biopsy. He comments at some length on the "regional indications" and states, in regard to tumors of the breast: "When a woman presents a lump or induration in the breast, one assumes a serious responsibility if he delays in making a positive diagnosis. The most direct method is to remove the mass and determine its nature at once."

It is generally agreed that in the presence of a tumor a biopsy is indicated if only as "a confession of the inaccuracy of clinical judgment," but it must be admitted that there are certain dangers associated with the surgical removal of such specimens. These dangers have been fully pointed out by Ewing, and need not be enlarged on here. In general, however, it may be stated that a positive and reasonably accurate diagnosis of any tumor must, of necessity, form the basis of effective and intelligent treatment and that a biopsy affords the simplest and surest method of attaining this end.

To the points which Ewing has made, we should add two more, applicable in particular to the less frequently employed but nonetheless important biopsies of bone tumors and lymph nodes.

For the proper diagnosis of the removed tissue, it is essential that the material be adequate in amount, satisfactorily fixed and properly stained. Nowhere is this more true than in the field of lymph-node diseases and bone tumors, yet again and again the biopsied tissue, often inadequate in amount, is allowed to dry and shrivel before fixation or is summarily dropped into an inadequate fixative in pieces too thick to allow of proper preservation. The resulting sections, often poorly cut and stained, cannot be properly diagnosed, and the pathologist is frequently forced to state: "Probably malignant tumor; exact nature uncertain." Surgeons expecting to do such biopsies can easily obtain directions from competent pathologists for the proper preservation of tissue; and he who expects to send a case to some large medical center for consultation or for treatment if the mass should prove to be malignant would often be far better served if biopsy were deferred until the patient is seen at such a medical center. Neither the patient nor the referring doctor nor the consultant loses by such a procedure.

In the field of lymph nodes, one further error is not infrequently committed. There is a tendency to pass by the large nodes and remove one which is small and perhaps more readily accessible. Not infrequently such a node is found to be merely the site of an inflammatory or hyperplastic process in no way indicative of the major underlying disease. The pathologist may be compelled to report: "No evidence of tumor formation"; nor can he justifiably modify this statement on the basis of the clinical findings. The histological evidence before him is clearly indefinite, through no fault of his own. He can, at best, suggest another biopsy, but

it is no easy matter to persuade a patient to undergo a second operation even it be a minor one and for his own good.

If a biopsy is done, let it be performed thoughtfully, skillfully and circumspectly, with full recognition of the impossibility of any accurate diagnosis from inadequate or poorly prepared material. Lack of attention to these details may — and often does — lead to disappointment, false statistical data and even danger.

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THE TUBERCULOSIS PHENOMENON

In this issue of the *Journal* is a discussion of the current programs for tuberculosis control. Many who have cultivated the habit of passing by these perennial presentations would do well to pause and read one now and then.

In the early days of the "war on tuberculosis" a great emotional stimulus was needed. Zealots were good promoters then, for they fostered the combative spirit and thus produced the action and reaction necessary to publicize and popularize the cause. At first they were mere voices, clamorous in the wilderness of a still medieval point of view toward sickness and its prevention. Gradually they began to achieve; they attracted more attention, more sympathy, more support, more acceptance of their efforts as one of the great moral movements of the day. About them there appeared the little parasitic enterprises and fads that always attach themselves to a successful venture, just as barnacles attach themselves to a mighty rock. The movement became stabilized and at length an accepted part of what is now recognized as a public-health responsibility.

No longer should we think of the "cause" or a "war." Such an emotional background is neither needed nor wanted by those who might equally resent being referred to as an "army" of workers. It is an established group of specialists, already divided into their own sub-specialties, paid largely from public funds and rendering their services largely in public institutions. They no longer need the zealots of the past. They go about their work in a deliberate, business-like manner. It makes little difference to them what kind of seals you buy at Christmas and whether you open your bedroom windows at night.

With a mortality reduction of approximately 70 per cent in the past forty years, what used to be called the tuberculosis problem not only seems well under control, but is itself already creating new problems. The momentum of eradication is greater than statisticians dared to predict a few years ago. It can easily be maintained if living standards can be maintained and early cases recognized. Within a few years the need for new beds for tuberculosis will disappear. Within a few generations the herd immunity of those who have encountered and resisted the disease will disappear. It is no longer a problem in the ordinary sense — it is a phenomenon.

OBITUARY

RICHARD CLARKE CABOT

1868-1939

In every generation there are restless souls, who cannot be made to fit the common mold. A few of these are valuable in keeping their communities and professions in a ferment by their constant challenge to the existing order of man's thought and action. But when, in addition to possessing these attributes, a rare individual is endowed with the divine fire and makes important contributions to the pioneering progress of humanity, then indeed we recognize a great leader. In the thick of the fray such recognition comes slowly but as the smoke of the battle clears the acclaim is universal.

Richard Clarke Cabot was born in Brookline, Massachusetts, May 21, 1868, and came within two weeks of living seventy-one years. He was the fifth son of James Elliot and Elizabeth (Dwight) Cabot. After preparing for college at Noble and Greenough School, he entered Harvard University from which he received his A.B. degree, summa cum laude, in 1889 and his M.D. in 1892. Three other degrees he received later in life in recognition of his attainments, an LL.D. (Rochester, New York, 1930), an L.H.D. (Syracuse, 1934) and a D.D. (Colby, 1938).

In 1894 he married Ella Lyman in Waltham

and spent the next half year traveling and studying in Europe before settling down to practice and to begin his teaching and researches at 190 Marlborough Street in Boston. His married life was an extraordinarily happy one and continued fortunately for forty years. The devotion of Dr. Cabot and his wife to each other was a constant joy to their friends and a source of inspiration for much of his philosophical writing.

It is not possible to divide Dr. Cabot's life into clear-cut periods of activity, for one interest merged into another gradually and naturally. In fact what some consider a late interest, namely philosophy, came early in life as shown by the facts that he was a lecturer in that subject at Josiah Royce's Harvard Seminary course in logic in 1903-1904 when but thirty-five years old and that in 1905 at thirty-seven he inaugurated social service at the Massachusetts General Hospital. It is true, however, that the earlier half of his professional life was preponderantly concerned with medicine and the later half with social ethics.

Twenty-five years ago when I presented a letter of introduction from him to a medical leader abroad he expressed surprise that Dr. Cabot could still be alive after such a record as he had already made. And yet he was then only forty-five years old.

The reason for Dr. Cabot's early fame in medicine was his great ability and industry in pioneer work. He was indefatigable to the end of his days and always a pioneer. At the age of twenty-eight, in 1896, he published his first book *Clinical Examination of the Blood*, which went into five editions in eight years. In 1899, at thirty-one, he published *Serum Diagnosis of Disease*. His service in the Spanish-American War on the hospital ship *Bay State* as lieutenant in the Medical Corps was a great stimulant to his increasing interest in the blood and in infectious diseases. But soon he left these fields, although he could easily have continued in them as a leading authority had he so chosen. He feared a narrowing of his viewpoint. In the ten years from 1901 to 1911 he wrote three medical books of wide interest, the first of which, *Physical Diagnosis*, has been used the world over as a textbook and has appeared in twelve editions from 1901 to 1938 (the last edition with Dr. F. Dennette Adams). The other two medical books published in that decade were *Case Histories in Medicine* (1906) and the first volume of *Differential Diagnosis* (1911); the latter went into four editions from 1911 to 1919 and was followed by the second volume, of which there were three editions from 1915 to 1924.

More important, however, than any of his books in that decade was his introduction of autopsy teaching in 1910, the first of the long and continuous series of his famous *Clinicopathological Conferences* that are still carried on actively at the Massachusetts General Hospital in a modified way. Dr. Cabot was an incomparable teacher, a master at this kind of exercise, always willing to commit himself to stimulate his students and his colleagues to learn through their errors as he did through his own.

During this period of intensive medical work, 1901 to 1911, Dr. Cabot also carried on a practice and served at the Harvard Medical School (appointed instructor in medicine in 1903 and assistant professor in 1908) as well as at the Massachusetts General Hospital (physician to the Out Patient Department in 1898 and assistant in medicine in 1899). From 1902 to 1929 he conducted very popular summer courses in internal medicine at the hospital. These were attended by physicians from all parts of the country and constituted pioneering work in the important but neglected field of postgraduate medical education in the United States.

It was still in that decade of his greatest medical activity that Dr. Cabot made his second vital contribution, in another though related field. In 1905 he started medical social service at the Massachusetts General Hospital and in 1909 wrote a book *Social Service and the Art of Healing*, of which there was a second edition in 1928.

In 1930, when a special Social Service Fund was raised for educational purposes in commemoration of the twenty-fifth anniversary of the beginning of hospital social service, Dr. Mary Lawson wrote:

> I am so glad that you asked me to give my bit to continue the wonderful work that Dr. Cabot started. I remember its beginning very well. I was with him at the time. Did you know that he had something of the kind in mind ever since he was a small boy of ten? I do not believe there is anyone more thoroughly acquainted than I am with the wonderful unselfishness of Dr. Cabot's character. He is always doing something for others. When he started the work he had to meet so much unbelief in its usefulness; but he has that wonderful gift of seeing beyond and he knew what its future would be. It started in a little corner of a corridor in the Out Patient Department surrounded by screens, with one paid worker and volunteers. Dr. Cabot consulted with and advised the helpers daily, being himself responsible for all financial aid beyond what little might be given by subscription. The work was Dr. Cabot in those days.

This tradition of service Dr. Cabot maintained to the end of his years, passing on in the course of time most of the responsibilities and a host of important developments in this social-service field to the able leader who has taken his place, with her own mantle as well as his, Ida M. Cannon. Miss Cannon has surrounded herself with other

workers still pioneering in social service within and without the walls of the old hospital. All parts of the world have followed the example set by this group, just as they have studied Cabot's *Physical Diagnosis* and *Clinicopathological Conferences* and recognized his epoch-making medical contribution of 1914.

During the second decade of the century Dr. Cabot continued his intensive work in medicine and social service, adapting it from 1917 to 1919 to suit the changed conditions of the days of the World War, when he served as chief (major to lieutenant colonel, Medical Corps) of the medical staff of Base Hospital No. 6 (Massachusetts General Hospital Unit) in the A. E. F. in France, and under the Red Cross. established dispensaries for refugees and lectured in French on social work at the Collège de France. In 1912 he was appointed as one of the two chiefs of the medical staff at the Massachusetts General Hospital, which position he held till his retirement in 1921, and in 1919 he was appointed professor of clinical medicine and professor of social ethics at Harvard, not a whole settee as in ancient days but twice as many chairs as has been possible for the average mortal to occupy in our own generation.

It was in 1914, however, just at the beginning of the World War, that Dr. Cabot made his most important medical contribution in a short but vital paper published in the *Journal of the American Medical Association*, a landmark in medical history, which places him as the greatest contributor to cardiology in our generation. The war undoubtedly obscured this advance,—as it did that other very important American contribution to cardiology by Dr. James Herrick, of Chicago, on coronary thrombosis in 1912,—but now, after twenty-five years we can appreciate its full significance, and Dr. Cabot's name becomes associated with that of Sir James Mackenzie as a prime leader in the field of heart disease in the past half century. Yet he made this significant contribution merely in passing, as it were. Although heart disease was one of his pet hobbies in medicine,—witness his volume *Facts on the Heart* (1926),—it was a lesser interest after all, and he never pretended to be a heart specialist.

To some who are not cognizant of the great advances in cardiology since the World War this contribution of Dr. Cabot's in 1914 may still be news, but to those of us who have concentrated in the field it is the foundation of much if not most of our work in the last two decades. The paper was entitled "The Four Common Types of Heart Disease." For the first time, proper emphasis was laid on the etiologic diagnosis of heart disease, in contrast to the overemphasis of structural defects that had been current for over two hundred years. The revolution in point of view has been amazing. Where at one time, in fact for generations, textbooks and papers had been preponderantly involved with such subjects as mitral regurgitation, myocarditis and pericarditis, they now present as a primary interest the causes of heart disease. Not only has such a viewpoint become of major importance in routine diagnosis, prognosis and treatment, but it has also stimulated the essential studies of the etiologic factors behind heart disease with much promise in the fundamental field of preventive medicine. Sir James Mackenzie's great service to cardiology was in setting forth clearly the need of paying attention to disorders of function of the heart; Dr. Cabot's contribution on etiology was even more significant.

It was during this same decade, 1910 to 1920, that Dr. Cabot pioneered in still another medical field and thereby aroused the ire of many physicians and medical societies. In his militant bluntness and precipitate desire to correct the errors of the practice of medicine he wrote to the laity on the subject of *Better Doctoring for Less Money*, many years before it became fashionable to belabor the family doctor or specialist. Many persons with less of the zeal of the impetuous reformer believe that there are gentler and perhaps wiser ways to correct the errors of today than by substituting the errors of tomorrow, but undoubtedly these stormy methods have a certain value in hastening the return to a sensible equilibrium after all the shouting and the tumult have subsided.

Another extracurricular activity of Dr. Cabot's took place for a few months prior to the entrance of the United States into the World War. Believing strongly in the need of our taking the side of England and France on the battlefield itself, he and his wife went West on a vigorous speaking tour to rouse the country and did not stop until war was declared. He could not sit still when something in which he had his heart needed to be done.

In completing this brief glimpse of Dr. Cabot's rich life, let us view the post-war years, the last two decades. After the stirring days of the quarter century following the beginning of his professional career this latter period became relatively peaceful, actually richer and riper. To some who did not sense this change it seemed almost as if he had retired. On the other hand he went on to higher levels, leaving to us others the study and the care of the body. With the years he came to realize with increasing force the need of the study and the care of the spiritual side of man, his character and personality, truly his soul.

Through the years by a process of natural evolution he came to be less interested in man's body or even in man's mind than he was in his soul. Even in the hard war years I can recall in many quiet conversations with him how impatient he was with the mere healing of wounds and illnesses, and the education to be found in most schools and universities. Morons can be superbly healthy, criminals can be highly educated. It was the spiritual quality of any individual that attracted his attention, and the possibility of its cultivation that became to him a challenge. Probably to most mortals this realization comes with force enough to demand attention only in advanced age when it is too late to act, but Dr. Cabot was still strong and full of years when he set to work to tackle these problems. This richest part of his life, too recent for us, is least known and appreciated. One of the studies inaugurated during the last few years — the Cambridge-Somerville Youth Study — will take ten more years to run.

Scant witness of Dr. Cabot's growing interest in human society and in man's soul is to be found in a number of books published in the last twenty-five years of his life: *What Men Live By* (1914); *Adventures on the Borderland of Ethics* (1926); *The Goal of Social Work* (1927); *The Meaning of Right and Wrong* (1933, 1936); *The Art of Ministering to the Sick* (with Reverend R. L. Dicks, 1936); *Christianity and Sex* (1937); and *Honesty* (1938). When he died he was at work, with a literary friend, on what he believed to be his most important philosophical treatise, a summing up of values in life.

I cannot forbear mention of Dr. Cabot's final illness, which lasted nearly a year after more than twenty years of bother with both peptic ulcers and angina pectoris (despite which he played tennis until nearly seventy). Uncomplaining and brave, he followed the course of his own heart failure with great interest, challenging our diagnoses if he thought we were off the track and helpfully too, and hastening to concede the value of various medicines as we gave them, completely wiping out any reputation he may have had earlier in life as a therapeutic nihilist. He always demanded to know the truth of what we thought about his condition and did not flinch from pain or from the realization of his grave prognosis. He persuaded us to allow him for his mind's sake to work on his books and to teach, even when he was an invalid. What he did in this way seemed to help him physically also, as he predicted. For months his classes came to his bedroom twice a week and cherished this privilege as they will the memory of it.

His death ends a life of great service to man. Had he accomplished what he did in any one of his three chosen fields — medicine, social service, ethics — it would have been more than enough.

Through his last will and testament his spirit will continue to be active for many years to come. With rare wisdom he has bequeathed in his wife's name a fund to be used by trustees whom he selected to foster the work of any individuals not otherwise adequately supported who give promise of making important contributions to humanity in any field of activity: art, music, literature, philosophy, theology, education or science. Causes and institutions merely as such he did not wish to help, but as they are related to individuals of promise they may be incidentally benefited.

To others I must leave an account of his interests and talents in music, dramatics, literature and religion — his sense of perfect pitch, his Christmas carol choruses on Beacon Hill, at the Massachusetts General Hospital and in France, his skill with the violin, his readings of poetry and prose to groups of friends, his acting in theatricals, his sermons and his Sunday School lessons. To one person above others, Alice O'Gorman, Dr. Cabot's secretary, I add a special word of tribute; without her constant and skillful aid, her quiet encouragement and her understanding care he could not have accomplished what he did.

To me, as to many others, Richard Cabot was always a stimulating counsellor, vigorous critic and warm-hearted friend. In his example he has left a great heritage.

P. D. W.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY*

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

INVERSION OF THE UTERUS

Inversion of the uterus is a rare complication of labor. The incidence varies in accordance with the author from 1 in 20,000 to 1 in 190,000 deliveries. It generally accompanies or follows the birth of the placenta and is more frequent in primiparas. The mortality as quoted in the literature is high, but with immediate recognition and proper treatment it should be low. It may be spontaneous or induced; it may be partial or complete. In the complete cases the fundus of

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section.

the uterus passes through the cervix and may even appear outside the vulva. The condition occurs chiefly in the presence of atony. First there is a dimpling or cupping of the fundus of the uterus, which may be due to the weight of a high-implanted placenta, to a forcible attempt to deliver a relaxed uterus or to traction on the cord. The fundus projecting into the uterine cavity acts as a foreign body and stimulates forcible contractions of the rest of the musculature in an attempt to expel it — hence the complete inversion.

Inversion of the uterus is generally accompanied by profound shock and profuse hemorrhage. The diagnosis should be easy. The fundus can no longer be felt above the symphysis. There is a large, rounded, spongy mass protruding from the cervix, associated with profuse bleeding and signs of collapse.

Successful treatment depends primarily on improving the condition of the patient before any measures to replace the uterus are undertaken. The profuse bleeding may generally be controlled temporarily by firm packing of the vagina; intravenous fluids and transfusions should be given. Then, and not until then, it is proper to attempt a gentle manual reposition of the uterus. At times this can be successfully accomplished under ether anesthesia; often, however, it will be found that the cervix has shut down so tightly that it is impossible to push the large boggy fundus through it.

Should this be the case, immediate operation of the type suggested by Huntington* is the method of choice. The patient is placed in Trendelenburg position, anesthetized and prepared. The abdomen is opened in the lower midline. A funnel-shaped depression at the site of the cervix is then evident from within the pelvis, with the tubes and round ligaments leading into the opening. The operator and his assistant, each armed with Allis forceps, then grasp the invaginated portion of the uterine wall about 2.5 cm. below the cervix and draw it gently upward. New bites are taken just below the original ones, and thus the fundus of the uterus is gradually returned to the abdominal cavity, that part which went through the cervix last being the first to be withdrawn.

If the uterus is not replaced, the patient may recover from the shock and hemorrhage and live to face the dangers of sepsis. Rarely there may be a spontaneous return of the uterus to the abdomen, but cases have been reported in which the cervix has shut down so completely that gangrene and sloughing of the fundus have occurred.

When untreated, the condition occasionally passes on to the chronic stage, with marked involution or atrophy of the fundus. In chronic cases in which the inversion has been present for more than a month it is usually impossible to replace the uterus without splitting the cervix by some form of vaginal operation.

This condition is of interest in Massachusetts because in 1937 there were two deaths from inverted uterus, and in 1938, one. Subsequent case reports in the *Journal* will deal with acute and chronic inversion of the uterus.

*Huntington, J. L.: Acute inversion of the uterus. Boston M. & S. J. 184:376-380, 1921.

LEGISLATIVE NOTES

The approved enabling act for medical insurance was presented to the Committee on Rules of the Massachusetts Legislature on June 8. No report had been made on it up to June 21. The proposed act reads as follows:

AN ACT ALLOWING THE INCORPORATION OF NON-PROFIT MEDICAL SERVICE CORPORATIONS.

Be it enacted by the Senate and House of Representatives in General Court assembled, and by the authority of the same, as follows:

Section 1. Any corporation organized under the laws of the Commonwealth for the purpose of establishing, maintaining and operating a non-profit medical service plan whereby the cost of certain medical care and service may be paid for in whole or in part by such corporation, to such of the public as become subscribers to said plan under a contract which entitles each subscriber to specified payments for the cost of certain medical care or service, shall be governed by this chapter and shall be exempt from all provisions of the insurance laws of the Commonwealth, except as otherwise provided in this chapter. Wherever the term "medical care and service" is used in this chapter it shall be deemed to include surgical care and service and the term physician shall also be deemed to include surgeon. The term medical care and service as used in this section shall be construed to be medical care and service as defined by the statutes of Massachusetts and the opinions of the Massachusetts Supreme Court.

Section 2. Persons desiring to form such a non-profit medical service corporation shall incorporate as provided in section three of chapter one hundred and eighty. At least a majority of the directors of such corporation shall be at all times persons licensed to practice medicine in the Commonwealth. Every certificate of organization of a corporation subject to this chapter filed under said chapter one hundred and eighty shall have endorsed thereon or attached thereto the consent of the Commissioner of Insurance and of the Commissioner of Public Welfare.

Section 3. Any corporation subject to this chapter may enter into contracts with persons, to be known as "subscribers," calling for the payment by such corporation, subject to such limitation and at such rates as shall be specified in such contracts, of the cost of medical care furnished to such subscriber, or to his or her dependents or family, by a physician or physicians licensed to practice medicine. All such contracts with subscribers shall be in writing and the terms and provisions thereof shall at all times be subject to the approval of the Commissioner of Insurance. The rates or bases at or upon which physicians are to be compensated for medical care or service rendered to or for the benefit of a subscriber shall

whenever the compensation of such physician is to be paid by such corporation, be approved by————————

Section 4. Every such corporation shall annually, on or before the first day of March, file in the office of the Commissioner of Insurance a statement, verified by at least two of the principal officers of said corporation, showing its condition as of the thirty-first day of December next preceding. Said statement shall be in such form and shall contain such other matters as the Commissioner of Insurance shall prescribe. A corporation neglecting to make and file its annual statement in the form and within the time specified shall forfeit one hundred dollars for each day during which such neglect continues, and, upon notice by the commissioner to that effect, its authority to do new business shall cease while such default continues.

Section 5. The Commissioner of Insurance, any deputy or examiner, or any other person whom said commissioner shall designate, at least once in three years and whenever he deems it to be prudent, shall visit any such corporation and examine into its affairs, shall have free access to all the books, papers and documents of the corporation that relate to its business, and may summon as witnesses and examine under oath its officers, agents or employees, or other persons in relation to its affairs, transactions and condition. The commissioner shall require every such corporation to keep its books, records, accounts and vouchers in such manner that he or his authorized representatives may readily verify its annual statements and ascertain whether the corporation has complied with the law.

Section 6. All acquisition costs in connection with the solicitation of subscribers to such medical service plans shall at all times be subject to the approval of the Commissioner of Insurance.

Section 7. The funds of any corporation subject to this chapter shall be invested only in securities permitted by the laws of the Commonwealth for the investment of the capital of life insurance companies. The directors or other officers of such a corporation making or authorizing an investment or loan not included in the securities permitted for investment shall be personally liable to the corporation for any loss caused thereby.

Section 8. If the Commissioner of Insurance is satisfied that any corporation subject to this chapter has failed to comply with the provisions of its charter; or is being operated for profit; or is fraudulently conducted; or if said commissioner is satisfied that its condition is such as to render its further transaction of business hazardous to the public or to its subscribers; or if, in the opinion of such commissioner, (a) the officers and agents of such corporation have refused to submit to an examination under section five; or (b) such corporation has exceeded its powers or violated any provision of law; or (c) it has attempted to compromise with its creditors (other than physicians) on the ground that it is financially unable to pay its claims in full or is attempting so to do; or (d) it is insolvent; then, in any such case, he may apply to the Supreme Judicial Court for an injunction restraining it from further proceeding with its business in whole or in part. The court may forthwith issue a temporary injunction restraining the corporation from further transacting any business, and it may, after a full hearing, make the injunction permanent, and appoint one or more receivers to take possession of the books, papers, moneys and other assets of the corporation, and settle its affairs, and distribute its funds to those entitled thereto, subject to such rules and orders as the court may prescribe.

Section 9. Every corporation subject to this chapter is hereby declared to be a charitable and benevolent corporation, and its property shall be exempt from state, county, district and municipal taxes.

Section 10. No corporation subject to this chapter shall pay any salary, compensation or emolument to any officer, trustee or director thereof, as such, nor shall it pay any salary, compensation or emolument amounting in any year to more than $5,000 to any person, unless such payment be first authorized by a vote of its board of directors; provided, however, that such limitation shall not be applicable to the compensation paid to a physician for medical care and service rendered to subscribers. No corporation subject to this chapter shall make any agreement with any of its officers, trustees or employees whereby it agrees that for any services rendered or to be rendered to the corporation he shall receive any salary, compensation or emolument that will extend beyond a period of three years from the date of such agreement.

CHARLES C. LUND, M.D., *Chairman*,
Committee on State and
National Legislation.

DEATHS

ALLISON — CARL E. ALLISON, M.D., of Wakefield, died June 9. He was in his fifty-second year.

Dr. Allison received his degree from the Tufts College Medical School in 1914. He was a member of the Massachusetts Medical Society and the American Medical Association, and had practiced for twenty years in Wakefield.

His widow and a son survive him.

BERG — TEKLA A. J. BERG, M.D., of 109 Broad Street, Lynn, died June 8. She was in her seventy-first year.

Born in Ostersund, Sweden, she came to the United States when she was twenty-five years old and received her degree from the Tufts College Medical School in 1898. A member of the Massachusetts Medical Society and the American Medical Association, she was one of Lynn's oldest women physicians, having practiced there for forty-one years.

A brother, Dr. Ernest J. Berg, professor of electrical engineering at Union College, Schenectady, New York, survives her.

DONOGHUE — JOHN J. DONOGHUE, M.D., of Worcester, died March 21. He was in his sixty-fifth year.

Dr. Donoghue received his degree from the University of Michigan Department of Medicine and Surgery, in 1904. He was a former member of the Massachusetts Medical Society.

GAYLORD — JAMES F. GAYLORD, M.D., of Springfield, died June 8. He was in his fifty-first year.

Born in South Hadley, he received his early education there. He attended Dartmouth College and received his degree from the Dartmouth Medical School in 1914. He was a fellow of the Massachusetts Medical Society and the American Medical Association and was also a member of the Springfield Medical Association of which he was a former president, and the Alpha Kappa Kappa Medical Society.

His widow, two sons, a daughter, a brother and three sisters survive him.

GIBSON — DAVID H. GIBSON, M.D., of 115 Mt. Auburn Street, Cambridge, died June 8. He was in his fifty-fifth year.

After attending Cambridge Latin School he received his degree from Tufts College Medical School in 1921. Dr. Gibson was a member of the Massachusetts Medical Society and the American Medical Association.

His brother survives him.

REYNOLDS — GEORGE P. REYNOLDS, M.D., of Brookline, died June 6. He was in his forty-second year.

He graduated from Milton Academy in 1915 and spent the following year at Phillips Andover Academy. He received his degree from Harvard College in 1920 and from the Harvard Medical School in 1924, and interned at the Massachusetts General Hospital. After serving as assistant medical resident at Johns Hopkins Hospital he became assistant in medicine on the staff of the Massachusetts General Hospital and was appointed junior visiting physician on the staff of the Boston City Hospital in 1928. Dr. Reynolds was assistant in medicine at the Harvard Medical School from 1928 to 1935 when he was appointed instructor in medicine, which position he held at the time of his death. He was particularly interested in medical social-service work and in the application of practical psychological principles to the care of patients, and had contributed several articles on both topics.

In September, 1934, he was made physician to Milton Academy. Dr. Reynolds joined the Massachusetts Medical Society in 1927, serving as secretary to the Suffolk District Medical Society from 1933 to 1935. He was a member of the Council of the Massachusetts Medical Society from 1933 to 1937, being on the Committee of Arrangements from 1929 to 1934 and chairman of that committee in 1933. Dr. Reynolds was a fellow of the American Medical Association.

His widow, three daughters, his mother and a brother survive him.

NEW HAMPSHIRE MEDICAL SOCIETY

HARRIMAN — ALPHA H. HARRIMAN, M.D., of Laconia, died on May 30, after a long illness.

He was born in Albany, New Hampshire, on October 14, 1857, the son of Nathaniel and Rhoda A. Harriman. He was graduated from Bowdoin Medical College in 1883 and moved shortly to Sandwich, where he remained for three years before going to Laconia in 1887.

Dr. Harriman held memberships in the New Hampshire and Winnipesaukee medical societies.

Survivors are his widow, Mrs. Alice S. Harriman, a son, N. Joy Harriman, a daughter, Mrs. Murray W. Wright, of Nashua, and four grandchildren.

MACLEAY — ALFRED A. MACLEAY, M.D., aged sixty-nine, died Thursday, June 1, at the Notre Dame Hospital in Manchester after a long illness. Dr. Macleay, a native of Canada, came to Manchester from London, England, in 1899 and practiced as an eye, ear, nose and throat specialist until he retired because of ill health in 1932.

He was born in Castlebar, Quebec, October 8, 1869, the son of Alexander Monroe and Rosanna (Riddle) Macleay. He prepared for college at St. Francis School, Richmond, Quebec, and attended McGill University and Medical School, receiving the degrees of bachelor of arts, doctor of medicine and master of surgery.

Dr. Macleay was a life member of the British Ophthalmic Hospital in London, an honor granted him in recognition of his services there. He also was a member of the Canadian College of Medicine, the American Medical Association, the American Surgical Association, the New Hampshire Surgeons' Club and the state, county and local medical associations.

Besides his widow, the deceased is survived by a daughter, Mrs. Margaret Macleay Leavitt, a brother, Roderick Macleay, of High River, Alberta, and a sister, Mrs. John A. Wadleigh, of Danville, Quebec.

TIBBETTS — GUY D. TIBBETTS, M.D., aged fifty-one, died at Antrim, New Hampshire, on June 2.

He was born in Gloucester, Massachusetts, and was graduated from Tufts College Medical School in 1911. Dr. Tibbetts was one of 1000 American doctors loaned the British Government during the World War and for his services was awarded the Distinguished Service Cross.

He was a member of the Reserve Officers Corps, with the title of major, the Honorary Medical Society of Tufts College, the American Medical Association, the Hillsboro County Medical Association and the New Hampshire Medical Society.

MISCELLANY

HONORARY DEGREES AT BOSTON UNIVERSITY

Two doctors of medicine whose careers have been remarkable not only for services rendered but in length of time were presented honorary degrees in recognition of their achievements at the Boston University sixty-sixth annual commencement exercises held on June 12.

Drs. J. Emmons Briggs, of Boston, and William Otis Faxon, of Stoughton, were awarded the honorary degrees of Doctor of Science. Both are graduates of Boston University School of Medicine, and Dr. Faxon is the oldest living graduate of the medical school in active practice.

Dean Jesse B. Davis, of the School of Education, presented Dr. Briggs for the degree, and the latter was cited by Dr. Daniel L. Marsh as ". . . son of Boston University, whose superb abilities have been consecrated to the assuaging of human suffering and the advancement of medical science." Dr. Marsh's citation for Dr. Faxon, who was presented by Dean Alexander S. Begg, of the School of Medicine, was ". . . oldest living graduate of Boston University School of Medicine in active practice, for sixty-three years a glorious illustration of the finest connotation of 'the doctor in the home.'"

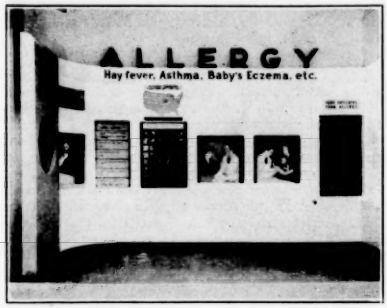
PNEUMONIA AND ALLERGY EXHIBITS AT THE NEW YORK WORLD'S FAIR

In the Medicine and Public Health Building, New York World's Fair, Lederle Laboratories are sponsoring the scientific exhibits on pneumonia and on allergy, each exhibit being controlled by a committee of eminent specialists on these diseases.

The pneumonia exhibit, surfaced entirely of white laminated "Beetle," occupies a booth 20 by 30 feet in a commanding position. It presents, pictorially, the best composite opinion of the medical profession on how a pneumonia case should be treated. The narrative is unfolded by means of a sequence of dioramas, pictures and charts. The story begins with an "animation" of a man walking in the rain, and takes him through typing and serum therapy and all the various progressive stages of a typical case of pneumonia to a final picture at the serum farm where his little daughter is pictured, saying, "Thanks,

old horse, you saved my daddy's life!" A "postscript" deals with sulfapyridine.

The exhibit on allergy tells, in changing dramatic sequences, three two-minute dramas of allergy: "Tommy Todd's Autumn 'Colds,'" "Mrs. Tucker's Wheezes" and



"Baby Bing's Eczema." By means of an animated question box and dioramas showing typical scenes in the doctor's office, a search for the offending allergic excitant in each of the three stories is conducted through information obtained by questions, scratch tests and an examination of the patient's family tree. An interesting part of the allergy exhibit is an illuminated transparency chart showing in full color, forty-eight of the most common allergic excitants. A separate series of little pictures invites the visitor to examine commonplace scenes for causes of allergy and then, by pressing buttons, to illuminate the concealed answers.

Physicians visiting the New York World's Fair are entitled to exclusive privileges to the Professional Club in the same building. Admission is obtained by simple identification as a doctor, without charge.

CORRESPONDENCE

ANNUAL DISCOURSE

To the Editor: Before drawing conclusions from my paper in Worcester regarding medical practice in Massachusetts, it is only fair to all concerned that those who did not hear me speak should read *all* that I said and not depend on abstracts which do not indicate the friendly, co-operative spirit in which the subject was presented. I understand that very shortly you are to print the entire paper, and I trust it will receive as much attention as the few excerpts which appeared in the press.

ELLIOTT P. JOSLIN, M.D.

81 Bay State Road,
Boston, Massachusetts.

REPORTS OF MEETINGS

GREATER BOSTON MEDICAL SOCIETY

A meeting of the Greater Boston Medical Society was held in the Beth Israel Hospital Auditorium on Tuesday, February 7. Dr. Louis M. Freedman presided and introduced the speaker of the evening, Dr. Soma Weiss, who spoke on "Etiological Factors and Therapeutic Measures in Circulatory Collapse and Shock."

Dr. Weiss stated that in the general field of diseases of the cardiovascular system, the three main disturbances are heart disease and congestive failure, arterial hypertension, and circulatory collapse. Of the three, the last is of the greatest importance and has been studied at the Thorndike Memorial Laboratory during the past eight years.

Circulatory collapse, or acute disintegration of the peripheral circulatory system, is by no means the same thing as congestive heart failure, and is commoner. The distinction between the two is important for therapeutic reasons; for example, in pulmonary edema with cardiac asthma venesection may be a life-saving measure, whereas in pulmonary edema with circulatory collapse, venesection may lead to death. However, the distinction must not suggest a too arbitrary segregation of the two conditions, for they may co-exist. Such is sometimes the case in diphtheria, for example, or in beri-beri, with an affection of both the heart and the peripheral vascular system. Certain drugs, such as the barbiturates, may also produce this picture. Even congestive heart failure itself may be complicated by peripheral circulatory collapse.

Peripheral circulatory collapse has been variously described as syncope, collapse and shock. The study of shock in animals and man is an old one. Dr. Weiss and his co-workers became interested in the hemodynamics of shock as a result of previous work in carotid-sinus syncope.

Dr. Weiss first discussed vasomotor reactions in normal subjects. The peripheral vascular system (of which the arterioles and venules are the most important elements) has as great a significance as the heart. Thus the capacity of an athlete depends not only on his having a good heart, but also on the state of response or "condition" of his peripheral vascular system, and the degree of economy with which it functions. In heart disease, the peripheral vascular system has great compensatory powers. Arteries, arterioles, capillaries, venules and veins all have their capacity for independent action, when tested with epinephrine, choline, histamine, nitrites, and so forth, and there is a wide variation in the response to these in each individual. This has an important bearing on the tendency to collapse or shock. Slides were shown, describing graphically the results of experiments, such as the effect of a loud noise or a deep breath. Epinephrine causes a rise in systolic pressure, a drop in diastolic pressure, and a rise in pulse rate; the plethysmograph recorded a rise in blood flow through the forearm but a drop in that of the hand and foot. This latter is explained on the basis that the forearm consists mostly of muscle and has relatively little skin.

Dr. Weiss next considered the disturbances of the vasomotor system. A healthy, normal individual watching a parade on a warm day might suddenly become weak and turn pale, with an ashen color and beads of perspiration, and finally, in from 10 to 20 seconds, might collapse and become unconscious. His arterial pulse will be weak or absent, the heart rate first rapid and then slow, the blood pressure low or indeterminable. This is "vaso-vagal syncope," a benign condition but nevertheless alarming.

In another case, a patient with severe, acute pancreatitis also might suddenly turn pale and ashen gray, with beads of perspiration on his forehead, a rise in heart rate, a drop in blood pressure, and a drop in pulse rate. This, lasting for 5 to 20 minutes is "circulatory collapse"—a serious syndrome. If this state lasts for several hours or even days, it is called "shock." Obviously there must be something in common between these three conditions.

In a particular study carried out by Dr. Weiss and his

associates, circulatory collapse was induced in the volunteer subject by the oral administration of 2 to 3 gr. of sodium nitrite. There was no or slight effect on the subject in the horizontal position but if he was suddenly tilted up to an angle of 75 or 90 degrees, syncope was an immediate result. If slowly tilted to 70 degrees, a state was obtained in which the systolic blood pressure stayed roughly at 60 or 70; consciousness was maintained but the mental horizon was definitely narrowed, the heart rate rose, and there was an ashen pallor and beaded perspiration. This state progressed gradually, as the blood pressure dropped, and the picture simulated exactly that commonly seen in certain infectious diseases and described in circulatory collapse. If sufficient time had elapsed, the experimental subject would have gone into a state of "shock." Therefore, one may postulate a common etiology for syncope, going on to collapse and then shock.

In all three states there is a disproportion between the circulating blood volume and the total vascular volume, there is a decrease in the return flow of blood to the heart, and also an acute or subacute cerebral anoxia. In ordinary syncope there is a temporary pooling of blood in the peripheral vascular system; in circulatory collapse the return is less rapid than in syncope; in shock, even if the balance is restored by one means or another, the patient will not return to normal for some time, perhaps never, or in other words, some permanent damage has occurred. Shock can be defined as a condition with a tendency to a irreversible process, whereas syncope and collapse are usually reversible.

In the literature, the terms "collapse" and "shock" are used interchangeably by many. There have been no very satisfying classifications, and the distinction between medical and surgical shock in particular is unwarranted.

From the point of view of treatment it is important to know whether collapse and shock are due (1) to the loss of some normal body constituent or (2) to the action of some substance produced in the body of some extrinsic agent. Treatment is dramatic, prognosis excellent in the first or the deficiency type; treatment is much more difficult and prognosis much poorer in the second or toxic type.

Dr. Weiss recapitulated by showing several charts, the results of his experiments. The decrease in return of blood flow may reach values of 30 to 40 per cent below normal before acute circulatory collapse supervenes, and even more if produced gradually. Vein stretchability is a definite factor in the pooling of peripheral blood. Epinephrine has no effect on collapse induced by nitrite, and pitressin accentuates the collapse.

A few special features of circulatory collapse and shock as regards treatment were presented. Dr. Weiss believes that too much emphasis has been placed on watching the course of arterial blood pressure in cases where collapse is a possibility. The arterial pressure is not a measure of blood flow. Thus, due to arteriolar constriction, the blood pressure may be normal but the flow very poor. On the other hand, when there is arteriolar dilatation, the blood pressure drops and the pulse rate falls, and yet no fainting occurs because the blood flow is good. Adrenalin thus may have little effect on blood pressure as compared with its marked action on the rate of blood flow; the clinical picture is one of anxiety, pallor and perspiration, and is typical of circulatory collapse. The diagnosis of collapse is warranted, in spite of the fact that the arterial pressure is normal. Certain persons may maintain a low pulse in the presence of collapse because their controlling-vagal mechanism is peculiarly sensitive. Anesthesia collapse is probably due in part to an increased tendency to collapse depending on the nature of the specific clinical condition, on fear, debility, and so forth. Superimposed on this there is the intrinsic effect of the anesthetic and of the surgical trauma. Cold is a factor in collapse, as in diabetic coma. The suddenness with which collapse comes on is explained on a basis of high activity of the reserve peripheral mechanisms; these reserves maintain normal balance for a definite length of time.

Recent efforts have been directed toward determining the degree of predisposition to collapse and shock in normal persons. One has not been able to foretell such a tendency, since powerful longshoremen fall victims as readily as do asthenic individuals. Certain tests with histamine have been used preoperatively abroad, but their value is still doubtful. There are certain general predisposing factors: (1) the patient's mental state; (2) a low blood sugar, or starvation; (3) fear, which is very significant in explaining certain serum reactions; (4) the heavy-set, plethoric individual; (5) advancing age; (6) sex.

Dr. Weiss presented a summary chart of measures for prevention and treatment. Specifically, deficiencies are to be supplied, such as water, salts, blood, oxygen, protein, glucose, "cortin," heat, and certain vitamins, such as vitamin B_1 in beri-beri; abnormal factors are to be eliminated or remedied, such as sleeplessness, fear, pain, "toxins" and chemical poisons. Symptomatically, the following measures are instituted to improve the circulation in general: shock position; heat or cold; blood transfusion; the administration of glucose, sucrose or gum acacia; sodium bicarbonate for the acidosis which usually develops. The value of drugs, such as strychnine, caffeine, coramine, cardiazol, ephedrine, and amphetamine (Benzedrine), epinephrine, synephrine, paredrinol and pitressin, was discussed.

The discussion of Dr. Weiss's paper was opened by Dr. Herrman L. Blumgart. The distinction between congestive heart failure and peripheral circulatory collapse is that in the former the veins are engorged, in the latter they are empty. He and Dr. Altschule had found that it took more than 20 cc. of physiological saline or 5 per cent glucose solution per minute intravenously to increase the work of the heart up to 100 per cent. In treating collapse, then, one should give 40 to 50 cc. of fluid per minute, but with careful attention to the patient's response. They also found that amphetamine raised the blood pressure without increasing cardiac output, as does epinephrine. Recent postmortem research has demonstrated conclusively that collapse causes changes in certain organs as a result of thrombosis of the vessels.

Dr. Charles G. Mixter discussed the subject of surgical shock. Primary shock is rare at present, being largely due to industrial or automobile accidents. Secondary shock is relatively common and is distinguished by not coming on until about forty-eight hours after the original injury. Whereas primary shock is best treated by immediate operation, secondary shock does much better after long delay, — of days, if necessary, — during which time the patient is reassured and his physiological balance restored as much as possible. The surgeon then must choose a proper form of anesthesia, induce good sedation, and see to it that the necessary amount of heat is supplied and that the head is kept in a lowered position. The incision should be wide so as to forestall pulling and stretching of structures.

Dr. Jacob Fine brought up the question of primary "peritoneal" shock, and emphasized the importance of psychogenic factors in treatment and prevention.

Dr. Weiss answered several questions put to him by physicians in the audience. He had had no experience

with the application of tourniquets to the limbs in collapse but advised strongly against it. He said it is, of course, of benefit in cardiac asthma. As for alcohol in treatment, Dr. Weiss believes it should be more generally used since it is a good ready fuel and is also an analgesic. Hemoconcentration is of no value as an indicator of circulatory collapse, since there is no change except in the late stages. Likewise there are no changes in the chemical constituents of the blood except in late stages.

WILLIAM HARVEY SOCIETY

At a regular monthly meeting of the William Harvey Society of Tufts College Medical School on Friday, April 14, in the Beth Israel Hospital auditorium, the speaker was Dr. Philemon E. Truesdale, of Fall River. Dr. Truesdale chose as his subject: "Diaphragmatic Hernia."

The first part of the evening, Dr. Truesdale read a summarizing review of the symptomatology of diaphragmatic hernia. There is interference with three of the most important body functions: circulation, respiration and digestion. Symptoms are characterized by their diversity and number. So far as the circulation is concerned, there is interference with the venous return to the heart, and the heart is moderately or markedly displaced, with torsion of the great vessels. Sometimes these conditions give no symptoms, but at other times there may be all the symptoms of cardiac disease. As the lungs are encroached on, hoarseness, a dry cough, air hunger, cyanosis and collapse may develop, besides other manifestations of pulmonary disease. The disturbance of the gastrointestinal tract leads to even more and various symptoms. Such symptoms are usually provoked by food immediately after ingestion, as are the circulatory and respiratory manifestations. The picture of intestinal obstruction may develop as a result of constriction of the colon at the diaphragmatic stoma.

Dr. Truesdale made a distinction between eventration and hernia of the diaphragm. The former may be either congenital or acquired as the result of injury to or paralysis of the phrenic nerve. Both forms may co-exist.

Since the symptomatology is so complex, a differential diagnosis is often difficult until the barium or bismuth meal demonstrates the lesion by x-ray. A few cases were first diagnosed as tuberculosis of the lung. In acute pancreatitis the left diaphragm may be high, but the blood diastase is elevated, as it is not in eventration of the diaphragm. The symptoms may suggest heart disease quite strongly, or may lead to a diagnosis of enlargement of the thymus. In the very young, the cough may be mistaken for that of pertussis, or the gastrointestinal symptoms of obstruction may lead to a diagnosis of congenital atresia of the bowel. Gall-bladder disease in general gives pain at midnight or later and is usually associated with certain diets, whereas diaphragmatic hernia usually gives rise to pain in the daytime and after any sort of meal. The pain is also more constant than it is in peptic ulcer.

Dr. Truesdale continued to instruct and entertain for the remainder of the evening by going through his extensive series of cases of diaphragmatic hernia and by illustrating with lantern slides.

NOTICES

SOUTH END MEDICAL CLUB

The next meeting of the South End Medical Club will be held at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, June 27, at 12 o'clock noon. Dr. Oscar Auerbach, pathologist, Sea View Hospital, New York City, will speak on "Pathogenesis of Empyema in Chronic Pulmonary Tuberculosis" and will show lantern slides.

Physicians are cordially invited to attend.

JOHN B. HALL, M.D., *Secretary.*

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS, UNDER THE PROVISIONS OF THE SOCIAL SECURITY ACT

CLINIC	DATE	ORTHOPEDIC CONSULTANT
Haverhill	July 5	Arthur T. Legg
Lowell	July 7	Albert H. Brewster
Salem	July 10	Harold C. Bean
Gardner	July 11	Mark H. Rogers
Brockton	July 13	George W. Van Gorder
Pittsfield	July 17	Francis A. Slowick
Northampton	July 19	Garry deN. Hough, Jr.
Worcester	July 21	John W. O'Meara
Fall River	July 24	Eugene A. McCarthy
Hyannis	July 25	Paul L. Norton

SALEM HOSPITAL TUMOR CLINIC

There is to be a teaching Tumor Clinic at the Salem Hospital, June 30, at 9 a. m., to be presided over by Dr. Channing Simmons, of Boston. Interesting cases seen and treated in the clinic during the past several months will be presented and methods of treatment reviewed.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, JUNE 26

MONDAY, JUNE 26

National Tuberculosis Association. Hotel Statler, Boston.

TUESDAY, JUNE 27

National Tuberculosis Association. Hotel Statler, Boston.

*10 a. m. - 12:30 p. m. Boston Dispensary tumor clinic.

*12 m. South End Medical Club. Headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

WEDNESDAY, JUNE 28

National Tuberculosis Association. Hotel Statler, Boston.

THURSDAY, JUNE 29

National Tuberculosis Association. Hotel Statler, Boston.

FRIDAY, JUNE 30

*10 a. m. - 12:30 p. m. Boston Dispensary tumor clinic.

SATURDAY, JULY 1

*10 a. m. - 12 m. Staff rounds of the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.

JUNE 26-29 — National Tuberculosis Association. Page 897, issue of May 25.

JUNE 27 — South End Medical Club. Notice above.

JUNE 27-29 — Medical Library Association. Page 941, issue of June 1.

JUNE 29 — Pentucket Association of Physicians, 8:30 p. m., Hotel Whittier, 5 Washington Street, Haverhill.

JUNE 30 — Salem Hospital Tumor Clinic. Notice above.

AUGUST 30 - SEPTEMBER 2 — Seminar in Physical Therapy. Page 857, issue of May 18.

SEPTEMBER — Boston Psychoanalytic Institute. Page 450, issue of September 22.

SEPTEMBER 4-6 — Institute for the Consideration of the Blood and Blood-Forming Organs. Page 941, issue of June 1.

SEPTEMBER 5-8 — American Congress of Physical Therapy. Page 857, issue of May 18.

SEPTEMBER 11-15 — American Congress on Obstetrics and Gynecology. Page 938, issue of December 8.

SEPTEMBER 14-16 — Biological Photographic Association. Page 941, issue of June 1.

SEPTEMBER 15-28 — Pan-Pacific Surgical Association. Page 863, issue of November 24.

OCTOBER 23 - NOVEMBER 3 — New York Academy of Medicine. Page 977, issue of June 8.

FALL, 1939 — Temperature Symposium. Page 218, issue of February 2.

DECEMBER 2 — American Board of Obstetrics and Gynecology. Page 1019, issue of June 15.

MAY 14, 1940 — Pharmacopoeial Convention. Page 894, issue of May 25.

JUNE 7, 8, 9, 1940 — American Board of Obstetrics and Gynecology. Page 1019, issue of June 15.

BOOK REVIEWS

Human Gastric Secretion. Bengt Ihre. Acta Medica Scandinavica. Supplement 95. 226 pp. Stockholm, 1938.

Ihre has made careful quantitative studies of the enzymes, acidity and total chloride in the gastric juice of 24 normal and 70 pathologic cases. For this study he emphasized the importance of a choice of method in obtaining specimens which would prevent the loss of juice through the pylorus, the admixture of duodenal juice due to regurgitation in the stomach and the admixture of saliva. He used, therefore, two Rehfuss tubes combined in the double tube of Lagerlöf and Agren. With this tube in place he could obtain gastric and duodenal juices quantitatively and separately. The saliva was avoided by continuous suction from the mouth. The juices were collected by continuous suction and routinely fractionated in twenty-minute periods. In order to get gastric juice after both humoral and neural stimulation, both histamine and insulin were used separately in every case. Insulin hypoglycemia was shown to be an excellent vagal stimulant. The determinations of acidity and total chloride were done electrometrically, and determination of pepsin according to the method of Willstätter and Waldschmidt-Leitz.

Studying the primary acidity and the regulation of acidity in special experiments he obtained support for the theory of Pavlov of a high and constant primary acidity. He concludes that the regulation of acidity mainly takes place through back diffusion of hydrogen ions through the gastric wall in accordance with the theory of Teorell and through the secretion of mucus which acts as a diluter and to a slight degree as an acid-binder.

The normal material consists of healthy individuals showing the least possible effect of the acidity-reducing factors. It was found that this was the case in individuals in the twenties, while with increasing age the acidity-reducing factors become more and more effective and a strictly physiologic secretion gets more uncommon. The highest acidity and total-chloride values were found in the normal series, and here the rates of secretion showed comparatively greater variations than did the values for the acidity and the chlorides. The same acidity values were encountered in men and in women, but in women there was a tendency toward a lower rate of secretion and they showed hypersecretion less often than did men.

The study of the pathologic cases was limited to gastric and duodenal ulcers, chronic gastritis and finally pernicious anemia. All cases were studied from the clinical point of view and were subjected to gastroscopy. On the whole, individuals with gastric ulcers showed a normal rate of secretion, while in most of those with duodenal ulcers there was more or less marked hypersecretion. In chronic gastritis without ulcer the tendency was clearly toward hyposecretion. Higher degrees of acidity than normal were not observed, and he concludes that the concept of hyperacidity lacks actual foundation. The majority of the pathologic cases showed lowered acidity. This is due to the greater effect of the acid-reducing factors in the pathologically altered stomach, that is, increased back diffusion of acid due to alteration of the mucous membranes. A correlation between total chlorides and acidity was noted: when the acidity declined or disappeared the chlorides did not fall below 120 milliequivalents per liter. Lower values are unquestionably due to admixtures of saliva.

Pepsin secretion is greatly accelerated by the vagal stimulation occurring in insulin hypoglycemia. Usually the pepsin elimination then reaches values two to three times greater than those before the stimulation. He established upper and lower limits for normal pepsin secretion and the existence of hyperpepsinia and hypopepsinia. When the vagal tonus is altered, variations in pepsin elimination depend on the rate of flow of gastric juice. Histamine and caffeine, which stimulate the secretion of gastric juice but do not produce any change in vagal tonus, have no effect on pepsin production.

When studying the secretion of pepsin it is necessary to know the amount secreted during a certain period, for the mere concentration in a sample collected over a short period is of little value. In this respect pepsin differs from acidity, which is high when the flow of gastric juice is high while the pepsin concentration tends to drop under the same conditions. The behavior of pepsin resembles that of the enzymes in the pancreatic juice, while the gastric acidity acts like the bicarbonate in the pancreatic juice. In this study sixty-minute collections were used to evaluate pepsin secretion.

Since histamine has no effect on pepsin production, it was only from vagal stimulation that the pathologic deviations in pepsin secretion became fully apparent. A great number of patients with gastric and duodenal ulcers and gastritis without ulcer showed pepsin secretion within normal limits. Different degrees of hypopepsinia were common features in gastritis, particularly in women; it was much less common in ulcers. Hyperpepsinia and hypersecretion may occur independently of each other; they are, however, usually correlated although not in the sense of cause and effect. They were present simultaneously in chronic ulcers of the stomach and duodenum, particularly in the latter. Hyperpepsinia, like hypersecretion, was less common in women than in men.

Trauma and Internal Disease: A basis for medical and legal evaluation of the etiology — pathology — clinical processes — following injury. Frank W. Spicer. 593 pp. Philadelphia, London and Montreal: J. B. Lippincott Co., 1939. $7.00.

Dr. Spicer states in his preface that this study "was prompted by the many problems which arise daily in the various courts of law relative to the causation or aggravation of disease by injury." He disclaims any intention of writing a book on traumatic surgery, but observes that "the more that is known of the pathological processes following injury the more intelligent will be the adjustment of claims." His primary object, then, is to aid those concerned with such adjustment by supplying an authoritative book of reference, and this has been accomplished by a compilation of cases and of the opinions of authorities from the literature, chiefly English and American. The twenty-five chapters deal with injuries classified according to the physiological system and the anatomical region involved, with the addition of special sections dealing with the relation of trauma to particular diseases, notably tuberculosis, peptic ulcer, appendicitis, diabetes, exophthalmic goiter, leukemia, arthritis, syphilis, tumors and electrical

injuries. Penetrating wounds and fractures, being manifest in their effects, are spoken of but briefly for the sake of thoroughness. Each chapter is thoroughly documented by references, and an adequate author and subject index is supplied.

Any observer of the contemporary scene will agree that the prevailing viewpoint is that any illness, accident, misery or unhappiness is the fault of society and should receive material compensation — from financial heart-balm for unrequited affections, through physical trauma, to allegedly libelous injuries to reputations. In the field of trauma, it would appear that Dr. Spicer has collected evidence to show that it may be responsible for practically every ill that flesh is heir to. He defines trauma as "a single or repeated mechanical injury resulting in contusion, crushing or laceration of the tissues" (Ewing), but apparently accepts psychic trauma, as for instance in exophthalmic goiter. With our present knowledge, imperfect though it is, of the profound influence of the endocrine glands, of the hormones and of the sympathetic nervous system on the circulation and on cellular metabolism, it would be unwise to deny the possible effect on disease of trauma acting through these agencies, but some of the author's examples of the responsibility of trauma seem farfetched. A few examples may be pertinent. A man falls 5 feet and lands on his sacrum, has brief unconsciousness, complains of pain in the neck and at autopsy, twenty-two days later, there is demonstrated acute epidemic encephalitis; the injury is adjudged to be the cause (p. 52). Is it not more likely that the intracerebral lesions of the incipient disease caused the fall? Narcolepsy may be the result of an injury antecedent by "several years" (p. 82). Torsion and gangrene of the omentum may be due to the exertion of putting up storm windows (p. 290). Peptic ulcer may be caused by a contusion: "occasionally the violence involves a portion of the body distant from the abdomen, the indirect forces acting along the principles of contrecoup" (p. 306). Quoting Osler and Kelly, "Trauma plays a definite role in acute appendicitis, . . . the length of time elapsing between the injury and the first symptoms being from none to two years" (p. 366). "Trauma may injure the graafian follicle or corpus luteum, without gross injury to the pelvic organs, sufficiently to cause many menstrual disorders" (p. 430). "Falls upon the feet may cause diabetes" (p. 442).

This reviewer does not mean to suggest by these examples that the book is not written with sincerity, but it seems fair to say that Dr. Spicer is presenting in the affirmative the case for the responsibility of trauma in causing internal disease. There are very few, if any, citations of authorities for the negative: perhaps such are not to be found in the literature! Dr. Spicer rarely presents his own opinion except as it may be inferred from his summaries of quoted authorities. This book should be an invaluable source book for physicians, patients, attorneys, clients, courts, industrial commissions and insurance companies. To the ten million persons who are said to suffer accidents in the United States every year, it will offer encouragement and aid in obtaining pecuniary relief in carrying the burden of illness. If conscientiously employed it will doubtless be a factor in promoting the just obligations of society to its members.

Industrial Surgery: Principles, problems and practice. Willis W. Lasher. 452 pp. New York: Paul B. Hoeber, Inc., 1938. $6.00.

Prof. Lasher brings together in the earlier chapters the ideal setup for the handling of industrial cases requiring surgical treatment. He advocates a unit equipped according to the size of the industrial plant and the nature of the injuries to which the employees are most liable, and a personnel adequate to give the service necessary. Typical forms on which careful records are to be kept are illustrated.

After consideration of general surgical conditions, the regional method is followed, beginning with finger and hand injuries and continuing with the other parts of the body, including a consideration of peripheral nerve injuries, bursas, fracture-dislocations, and so forth. The New York Labor Board's method of assessing losses as a basis for compensation for the various injuries is outlined. A number of admirable suggestions are made to aid in distinguishing between the three types of malingerers which the author recognizes. Illustrative cases are cited to emphasize points he wishes to bring out.

Though necessarily brief, the recommendations for treatment are clearly given and are based on the experience the author has had over a number of years as the director of the Employers' Liability Assurance Corporation, of New York. As befits an official in that position he probably would be classed as an ultraconservative. In certain subjects, like that of hallux valgus, for example, his opportunity to follow the surgical treatment of that deformity in industrial cases has not perhaps been extensive, accounting for his advocacy of surgical measures, in cases deemed suitable for operative treatment, that are not generally employed and for a reason not commonly advanced. In discussing the diagnosis of knee-joint lesions he records his experience with a method of examination which is all too little employed in this country, namely inflation of the joint with air to aid in bringing into high relief structures not otherwise clearly seen in an x-ray film. The same method is followed in joints of the lower extremity, the spine and the pelvic girdle. The last few chapters he devotes to such matters as hernias, cranial and facial injuries, those of the abdominal and thoracic viscera, the jaw and the neck and a miscellaneous group. In an appendix, a well-illustrated section is devoted to describing splints and certain tools commonly used in handling industrial accident work.

The book will be found very helpful to anyone engaged in this type of surgery and indeed it is worth having on the shelves of any practitioner's library.

Pediatric Symptomatology and Differential Diagnosis. Sanford Blum. 500 pp. Philadelphia: F. A. Davis Co., 1938. $5.00.

A reviewer — unless he be an utter misanthrope — is always pained to find a book of which he can say not one kind thing, but he is so circumstanced here. A treatise of this sort, to be worth its salt, ought to be both up to date and thoroughly comprehensive in scope, and this is neither. One might almost suspect, indeed, that it had been held in manuscript since about 1913, so far do its matters and, conspicuously, its point of view lag behind the times. As a test for its degree of obsolescence, let anyone turn, for example, to the sections on pneumonia and tuberculosis. For the rest, there are many subjects omitted altogether, such as renal rickets, oxycephaly, osteopetrosis, Schüller-Christian's disease, acrodynia, erythroblastic anemia, undulant fever, nephrosis and trichinosis. Granted that these are all uncommon conditions, it is precisely the uncommon that is most likely to send a man to a specialized volume on diagnosis.

There are many books, of course, which would be the better for some revision, but this is so faulty in all respects as to convince one that no amount of revision could ever make it useful.